Analyzing the Clinical outcomes, and timely interventions for Abdominal compartment syndrome among high risk individuals in a tertiary care hospital in India.

Dissertation submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the degree of

M.S. GENERAL SURGERY

Branch- I

PSG INSTITUTE OF MEDICAL SCIENCES AND RESEARCH

DEPARTMENT OF GENERAL SURGERY

APRIL 2013

CERTIFICATE

This is to certify that **Dr.K.Rajavel**, postgraduate student (2010-2013) in the department of General Surgery, PSG Institute of medical sciences and research, Coimbatore, has done this dissertation titled "***Analyzing the Clinical outcomes, and timely interventions for Abdominal compartment syndrome among high risk individuals in a tertiary care hospital in India***" under the direct guidance and supervision of guide **Dr.S.Premkumar** in partial fulfillment of the regulations laid down by The Tamilnadu Dr.M.G.R. Medical University, Chennai, for M.S., Branch – I General Surgery degree examination.

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DECLARATION

I, K.Rajavel, solemnly declare that this dissertation "*Analyzing the Clinical outcomes, and timely interventions for Abdominal compartment syndrome among high risk individuals in a tertiary care hospital in India*" is a bonafide record of work done by me in the Department of General Surgery, PSG institute of Medical Sciences & Research, Coimbatore, under the guidance of Dr.S.Premkumar, Professor and Head of Surgery.

This dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfillment of the University regulations for the award of MS Degree (General Surgery) Branch-I, Examination to be held in April 2013.

Place: Coimbatore

Date: 26th December, 2012 (Dr.K.Rajavel)

ACKNOWLEDGEMENT

I wish to thank our Principal for having permitted me to conduct this study in this hospital.

I am ever so grateful to the HOD and my Professor Dr.S.Premkumar, Associate Professors Dr Rajesh Kumar and Dr Krishnakumar and the entire faculty of the Department of Surgery for the kind guidance, valuable advice, supervision and encouragement while doing this study.

My thanks are also to my colleagues, Interns and Staff Nurses for the considerable help extended to me.

I wish to place on record my gratitude to all the patients who have been an integral part of this study. I am indebted to them for their willing participation.

Most importantly i would like to thank my wife and family and friends for their support for completion of this study.

CONTENTS

1.Introduction

INTRODUCTION

Abdominal Compartment Syndrome has very much importance in surgical practice and in critical care because of its effects on multiple organ systems and as the patients of this syndrome are critically ill.

The most common high risk individuals for ACS are post laprotomy patients.

Laprotomy may be associated with raised intra abdominal pressure which is defined as intra-abdominal pressure higher than 12 mm of Hg and one of it's most dreaded complications is the Abdominal Compartment Syndrome.

Abdominal Compartment Syndrome (ACS) is defined as the sudden increase in the Intra-Abdominal pressure (IAP).Base line is usually '0' resulting in changes in respiration, haemodynamic stability, renal perfusion and cerebral perfusion[(2)].

After laparotomy intra-abdominal pressure increases up to 10 mm of Hg. The physiological changes are observed when IAP rises above 15 mm of Hg which is also termed as Intra Abdominal Hypertension (IAH)[(3)].

Raised intra-abdominal pressure leading to abdominal compartment syndrome is a highly under-recognised source of morbidity and mortality associated with laparotomy. Hence

intensive monitoring of intra-abdominal pressure as well as early and aggressive management of abdominal compartment

syndrome is essential to ensure a successful outcome in a patient undergoing laparotomy.

More recently, laparoscopy has brought-out the consequences of raised IAP.

This study is being undertaken to evaluate the impact of IAP on outcome in High risk individuals.

DEFINITION

Abdominal Compartment Syndrome is defined as "adverse physiological consequences that occur as a result of sudden increase in Intra Abdominal pressure and resolve following abdominal decompression".

INTRA ABDOMINAL HYPERTENSION

is defined as raised Intra Abdominal pressure above normal. Normal Intra Abdominal Pressure (IAP) is 0-5 mm Hg. Intra abdominal pressure varies with position, body habitat and activity. Intra abdominal Pressure is measured in cm of water or mm of Hg.

(1 cm of H2O (water) - 0.735 mm of Hg).

Intra abdominal pressure of between 3 to 10 mm Hg is commonly observed post operatively without adverse effects.

2. Aims and Objectives

AIMS AND OBJECTIVES

A Prospective analysis to identify the

1) Incidence of

    a. Intra Abdominal Hypertension(IAH)

    b. Abdominal Compartment Syndrome(ACS)

2) Etiology

3) Effects on morbidity to the patient

4) Timely medical and surgical interventions made among high risk patients in a tertiary care hospital in Coimbatore, India.

3. Review of Literature

REVIEW OF LITERATURE

The relationship between laparotomy and rise in intra abdominal pressure and it's effects on various organ systems have received ample attention since the 19th century.

The raised intra-abdominal pressure and its consequences over the various organ systems has been noted since 1863 when Marey and Bureau described the relationship between Intra-abdominal hypertension and respiratory function[4].

In 1870 Paul Bert published a volume on "Physiologie comparée de la respiration" showed experiments in anesthetized animals, measuring intra-thoracic and intra-abdominal pressures through catheters inserted in the trachea and rectum respectively and described elevation of IAP on inspiration and the descent of the diaphragm[5].

In 1911 Emerson demonstrated the effects of IAP over morbidity of cardiovascular system[5]

Thorington and Schmidt reviewed on urinary output changed with BP changes [7].

Bellis and Wangensteen demonstrated changes in venous flow in the abdomen and extremities associated with abdominal distention[8].

Ogilvie, demonstrated the need of laprotomy which was performed for a patient with burst abdomen, packed with cotton cloth and sutured over wound edges, and once wound granulated was allowed for wound to contract [9].

Gross showed the benefit of so-called “staged abdominal repair” in omphalocele, hence stressing the importance of avoiding tension Early experience with laparoscopy led to recognition of the adverse effects of pneumoperitoneum associated increase in IAP: Ivankovich et al described cardiovascular collapse during gynecological laparoscopy and studied the physiology of the phenomenon[19,20].

Lenz et al, studying cardiovascular changes during laparoscopy, pointed out the dangers of pneumoperitoneum in patients with cardiovascular dysfunction, anemia or hypovolemia[21].

Richardson and Trinkle studied hemodynamic and Pulmonary alterations with raised intra-abdominal pressure[22].

Kashtan et al rediscovered the hemodynamic effects of increased IAP[23].

Harman et al as well as Richards et al demonstrated how elevated IAP adversely affects renal function and how abdominal decompression improves it[24,25].

Le Roith et al studied the effects of increased IAP on plasma antidiuretic hormone levels[26].

However, the recognition of abdomen as a compartment and the concept of intra-abdominal hypertension resulting in Abdominal Compartment Syndrome (ACS) have only recently received attention. Korn and associates first used the term ACS in 1980s[6].

Smith et al reported reversal of postoperative anuria by decompressive laparotomy[27].

Barnes et al in 1985 studied cardiovascular responses to elevated IAP[28].

Caldwell and Ricotta measured changes in visceral blood flow[29].

Jacques and Lee reported improvement in renal perfusion after evacuation of retroperitoneal hematoma which was the cause for increased IAP[30].

It is only in later 1990s that the patho-physiological consequences of the increased intra abdominal pressure (IAP) and abdominal compartment syndrome have been recognized in a wide spectrum of surgical patients and treated aggressively.

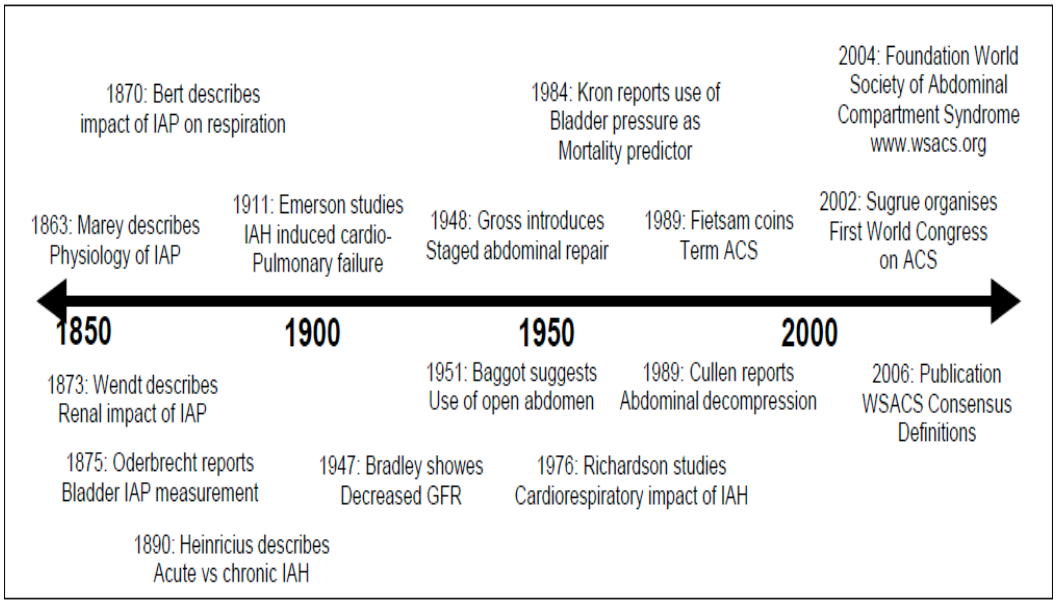
Since then most surgeon's started believing abdominal hypertension and the abdominal compartment syndrome as two different pathologic phenomenon which will have change in normal physiological process.

Two "collective reviews" of ACS appeared in 1995 and 1996 - opening the gate to numerous publications, recognizing IAHT/ACS in a large number of surgical intra abdominal and extra-abdominal, traumatic and non-traumatic scenarios—and providing an ever growing list of complications and consequences[33,34].

In 2004 the World Society of the Abdominal Compartment Syndrome (WSACS) was founded to promote research, foster education and improve the survival of patients with intra-abdominal hypertension and/or abdominal compartment syndrome.

Important landmarks in the history of IAH through the years 1850 to 2006 are summarised as follows :

(Chart 3. 1)



The reported incidence of Intra-Abdominal Hypertension and Abdominal Compartment Syndrome is about 32.1% and 4.2% respectively in the mixed intensive care unit population. In case of abdominal procedures it varies from 31.5-40.7%[36].Bruch JM et al[34] in 1996 graded intra-abdominal pressure using urinary bladder pressure as an indicator as follows:

Grade:	**Bladder pressure (mm of Hg):**
(1 mm of Hg = 1.36 cm of water)	
Grade 1	10 – 15

Grade 2	16 – 25
Grade 3	26 – 35
Grade 4	>35

The diagnosis of ACS depends on a very high degree of suspicion and recognition of patients at risk, identification of clinical syndrome and lastly measurement of IAP.

The various clinical parameters that are considered are abdominal distension, raised IAP above 20 mm of Hg, elevated peak airway pressure, massive I.V fluid requirements, oliguria progressing to anuria not responding to volume repletion, decreased cardiac output, hypoxemia refractory to increased PEEP, hypercapnia, wide pulse pressure and acidosis.

Acute abdominal hypertension can occur under the following clinical settings[37]:

• Peritonitis

•Severe abdominal traumatic injury

• Fluid overload

• Retroperitoneal hematoma

• Status post operative elective abdominal Surgeries

• Emergency abdominal surgeries

• Ishemic bowel and repercussion injury

•Acute Pancreatitis

• Intestinal Obstruction

•Mass per Abdomen

• Intra abdominal packing for bleeds (liver injury)

• Tension abdominal closure

• Ascites secondary to any cause

There are some well known risk factors associated with development of IAH and ACS[38]:

(Table 3.1)

Pathology	Aetiology	Antecedent cause	Aggravating factor
Fluid resuscitation Increased capillary leak	Massive resuscitation • Crystalloids • Colloids • Transfusions of blood or blood products Positive fluid balance Oliguria	Burns Trauma Sepsis Severe sepsis Septic shock	Acidosis (pH < 7.2) Coagulopathy Hypothermia
Increased abdominal contents	Liver disease with ascites Pancreatitis Peritoneal dialysis Peritonitis	Obesity causing increased mesenteric fat Laparoscopy- Pneumoperitoneum	Abdominal tumor Intraabdominal bleeding or tumor Intraabdominal abscess Retroperitoneal bleeding or tumor

Increased intraluminal contents	Gastric distension Gastroparesis Ileus Small bowel obstruction	Volvulus Ogilvie syndrome	Enteral feeding Intralumi nal tumor
Decreased abdominal wall compliance	Patient-ventilator dyssynchrony Increased work of breathing Extrinsic or intrinsic positive end-expiratory pressure Prone positioning	Burns with abdominal eschars Tight abdominal wall closures Abdomina l wall bleeding	Elevated BMI Central obesity Pregnancy Agitation/pain

Michel Chetham et al showed that Majchrzak in 2003 has classified abdominal compartment syndrome as [3]:

> "**Primary Abdominal cCompartment Syndrome** is essentially organ dysfunction and IAH in the presence of direct injury to the abdominal contents caused by trauma, peritonitis, ileus and haemorrhage.
>
> **Secondary Abdominal Compartment Syndrome** consists of elevated pressure and organ dysfunction caused by third space oedema and resuscitation. The examples are resuscitation of haemorrhagic shock patients and burns.
>
> **Recurrent Abdominal Compartment Syndrome** in which the patient has recovered from the ACS once but because of secondary insults the cycle begins again. This variety is associated with very high mortality rate"

The IAP is usually slightly elevated in the patient on mechanical ventilator support.

IAP increases in direct relation to body mass index of the patient.

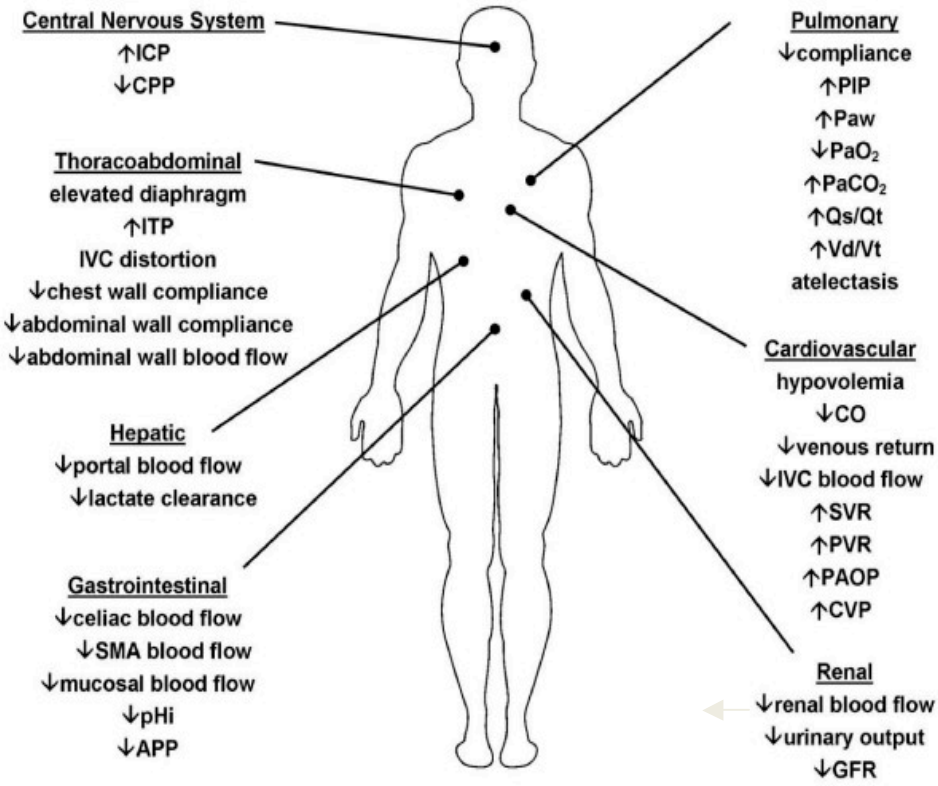
The compliance of the abdominal wall generally limits the raise in IAP but increases rapidly after a critical IAP.

Critical IAP varies from patient to patient, based on abdominal wall compliance.

The effects of intra-abdominal hypertension are not limited to the intra abdominal organs, but rather have an impact either directly or indirectly on every organ system in the body as shown in Figure 3.1.

Pathophysiologic Implications of Intra-abdominal Hypertension[39]

(Figure 3.1)



ICP – intracranial pressure; CPP – cerebral perfusion pressure; ITP – intrathoracic pressure; IVC – inferior vena cava; SMA – superior mesenteric artery; pHi – gastric intramuscosal pH; APP – abdominal perfusion pressure; PIP- peak inspiratory pressure; Paw – mean airway pressure; PaO_2 – oxygen tension; $PaCO_2$ – carbon dioxide tension; Qs/Qt – intrapulmonary shunt; Vd/Vt – pulmonary dead space ; CO – cardiac output; SVR – systemic vascular resistance; PVR – pulmonary vascular resistance; PAOP – pulmonary artery occlusion pressure; CVP – central venous pressure; GFR – glomerular filtration rate.

There are various systemic manifestations of raised intra-abdominal pressure as enumerated in the succeeding paragraphs[(2)]:

The **CNS** manifestations

- increase in the Intra cranial pressure and reduced cerebral perfusion pressure by reducing the cerebral perfusion pressure secondary to elevated intra thoracic pressure and elevated central venous pressure with reduced cerebral venous outflow.

The **CVS** effects

- include hypovolemia
- reduced cardiac output
- reduced venous return, raised Central Venous Pressure and raised Systemic Vascular Resistance

The effects on the **Respiratory system**

- raised intra-thoracic pressure,
- raised airway pressures,
- reduced compliance,
- reduced PaO2, raised PaCO2 and
- raised Shunt fraction.

The **Gastrointestinal system** reacts by

- reducing celiac blood flow,
- Superior Mesenteric Artery blood flow and Mucosal blood flow.

The renal effects manifest as reduced urinary output, reduced renal blood flow and reduced Glomerular Filtration Rate. Oliguria progressing to anuria and pre renal azotemia unresponsive to volume expansion is characteristic of renal dysfunction of ACS.

The **Hepatic manifestations** include

- reduced portal blood flow,
- reduced mitochondrial function and lactate clearance.

Abdominal wall shows reduced compliance and there is reduced rectus sheath blood flow. Increased pressure reduces the abdominal wall flow by 60% at an IAP of 10 mm of Hg or more. As collagen

deposits and resistance to infection are directly proportional to tissue perfusion and oxygenation, elevated IAP adversely affects the wound healing.

It is proved by several clinical studies and experimental studies that the adverse effects of Abdominal Compartment Syndrome are due to the mechanical factors influence on intra-abdominal, retroperitoneal and thoracic components.

Liberal intra-abdominal pressure measurement in the presence of known risk factors combined with implementation of an evolving and comprehensive resuscitation strategy have resulted in significant improvements in both short and long term outcome for patients who develop Intra-Abdominal Hypertension / Abdominal Compartment Syndrome[34].

Methods of Intra-Abdominal pressure monitoring:

IAP measured with direct and indirect methods. Though the direct methods are quite accurate over all ranges of Intra-Abdominal pressure, it is impractical and not feasible for routine practice.

Indirect pressure measurement is done through Inferior vena cava, gastric, rectal and urinary bladder pressure measurement.

The simplest and the method of choice is the **Urinary bladder pressure measurement.** However the measurement may be inaccurate in cases of neurogenic bladder, small contracted bladder and bladder trauma cases[40].

Rectal catheterization has the disadvantage of being uncomfortable to the patient and the need for the catheter to be 10cm above the anal verge failing which the values are not accurate.

Obeid and colleagues from Detroit found that with a standard 6mm Hg rise in IAP, as measured by an insufflator, it was best correlated with the intravesical measurements with a rise of 5.7mm of Hg. The gastric and rectal pressures were less reliable. He found that the gastric and rectal pressures were more position dependent and less reliable than the intravesical approach.

The most widely used method is the trans-urethral measurement of Urinary bladder pressure using a Foley's catheter. Kron et al first described this technique [39].

Sedrak et al had recently come out with a simple fluid column manometry system via the Foley catheter to measure the intra abdominal pressure [41]. By this simple method the pressure can be measured on an hourly basis.

A recent study indicated that although both the transducer technique and the catheter tubing method accurately reflected the intra abdominal pressure, the catheter method had a slightly stronger correlation between bladder pressure and the intra-abdominal pressure[42].

IAP measurement can be discontinued when the risk factors for IAH are resolved or the patient has no signs of acute organ dysfunction, and IAP values have fallen below 10-12 mmHg for 24-48 hours[43].

<u>IAP monitoring can be resorted to in the under mentioned conditions[35]:</u>

Sepsis / SIRS / Ischemia Reperfusion

- Sepsis and resuscitation with > 6 litres crystalloid/colloid or > 4 units blood in 8

hours

•Pancreatitis

•Peritnitis

•Ileus or bowel obstruction

•Mesenteric ischemia or necrosis

Visceral Compression / Reduction

•Large volume ascites/ peritoneal dialysis

•Retroperitoneal / abdominal wall bleeding

•Large abdominal tumor

•Laparotomy closed under tension

•Gastroschisis / Omphalocele

Surgical

•Intra-operative fluid balance > 6 litres

•Abdominal aortic aneurysm repair

Trauma

•Shock (ischemia-reperfusion)

•Damage Control Laparotomy

•Multiple trauma requiring resuscitation with > 6 liters crystalloid/colloid or > 4 units blood in 8 hours

•Major burns (> 25% TBSA)

MATERIALS AND METHODS

Type of Study: A Prospective Study.

Period of Study: Jan 2011 to July 2012.

4. Materials and Methods

Place of Study: PSG Hospitals, PSG IMS&R , Coimbatore, Tamil Nadu. 641004

Sample Size: 100 cases

Group A (Emergency cases) – 66 cases

Group B (Elective cases) – 34 cases

This study had been approved by the Institutional Ethical committee.

Plan of Study:

The detailed case history of the cases was recorded, clinical examination and investigations carried out as per the proforma enclosed.

Patients undergoing elective and emergency laparotomies were allotted under Group A and Group B respectively. Intra-Abdominal pressure was monitored daily till the IAP normalized or till post operative day 7 along with the pulse, blood pressure, respiratory rate, oxygen saturation, abdominal girth, urine output and arterial blood gas in patients who underwent a laparotomy.

All these factors were used to monitor the progress and assess the recovery of the patient.

Criteria for co-morbid conditions:

- Obesity: Patients with Body Mass Index (BMI) > 25 kg/m^2 were considered as obese

- Hypertension (HTN):
  - Patients who were known cases of HTN (on regular or irregular medication)
  - De novo detected cases of HTN (three consecutive blood pressure readings of 140/90 mm of Hg or more)

- Diabetes Mellitus (DM):
  - Patients who were known cases of DM (on regular or irregular medication)
  - De novo detected cases of DM (deranged BSL Profile)
    Normal values:
    BSL (fasting) 70-100 mg/dl
    BSL (Post Prandial) <140 mg/dl

Statistical analysis was performed using *SPSS* software.

Inclusion Criteria:

- All patients being planned for emergency and elective laparotomies.

Exclusion Criteria:

- Cases of Abdominal trauma
- Patients with pre-existing renal and hepatic derangement

Method of measurement of Intra-Abdominal pressure:-

A simple fluid column manometry system via the Foley's catheter was used to measure the intra-abdominal pressure.

The drainage tubing was marked along it's length and the Foley's catheter was marked as the Y-junction, which served as the zero reference point when it was at the level of the symphysis pubis.

The drainage tubing was marked at an increment of 1 cm on the tape, starting from the mark on Foley's catheter then 50 ml of sterile saline was introduced into the bladder.

After reconnecting the Foley's catheter to the drainage tubing, the zero reference point was taken at the level of symphysis pubis and the drainage tubing was raised vertically making sure that the transition from horizontal to vertical was at '0' mark and was not too abrupt. The distance the sterile saline raised vertically in the tubing was the intra-abdominal pressure in cm of H_2O. This was converted into pressure in mm of Hg by dividing the value by the conversion factor of 1.36.

IAP measurement was discontinued when the risk factors for IAH are resolved or the IAP values have been below 10-12 mmHg for 24-48 hours.

Measurement of Intra abdominal pressure

(Figure 4.1):

5. Results

Observations and Results

In our study, total 213 patients who underwent different surgeries were included. The study population consisted of 139 emergency cases and 74 elective cases. The study population was divided into two groups:

1. Group A: Patients undergoing Emergency Surgery (66 cases).
2. Group B: Patients undergoing Elective Surgery (34 cases).

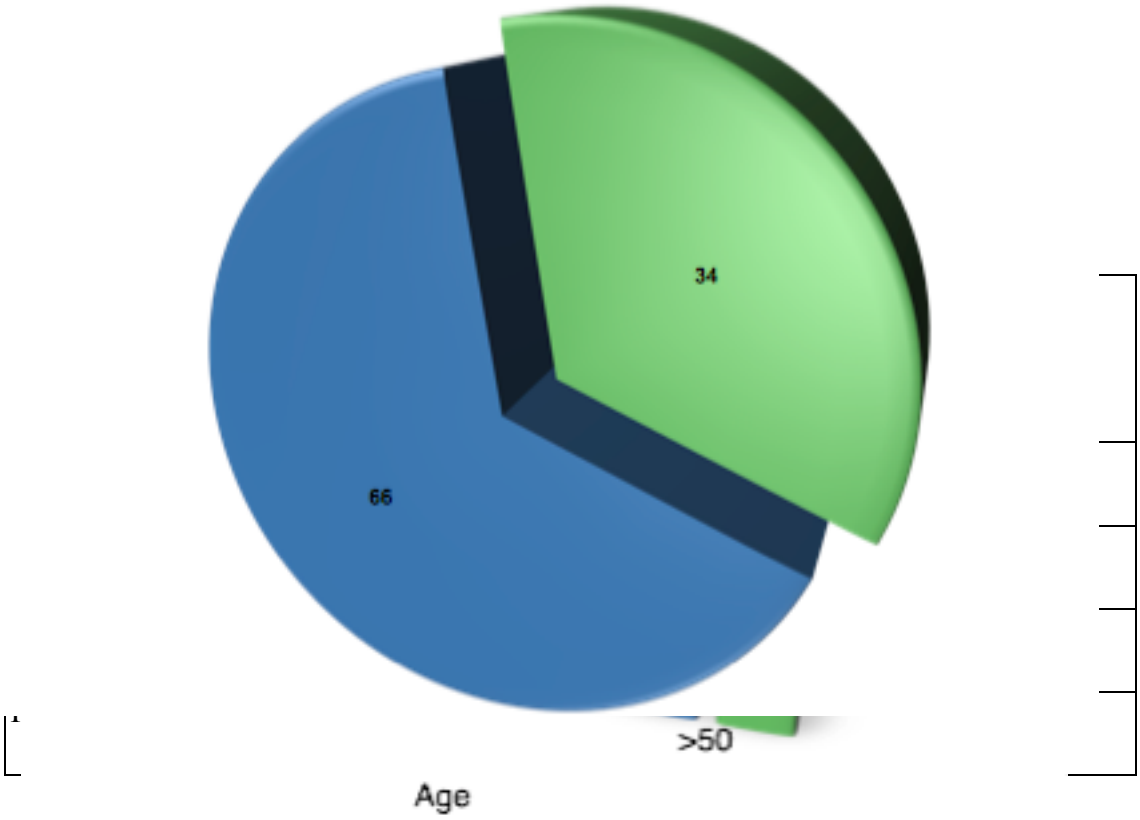
List of abbreviations Used:

- MAP: Mean arterial pressure
- RR: Respiratory Rate
- A/G: Abdominal Girth
- U/O: Urine Output
- SpO_2: Oxygen saturation
- TLC: Total Leucocyte Count
- BUN: Blood Urea Nitrogen
- ALT: Alanine Transaminase
- ALP: Alkaline Phosphatase

Table 5.1: Distribution of cases in study group according to Age and Gender:

Graph5.1

Pie diagram showing distribution of cases in study group according to Nature of Surgery



66.2% of the patients were male and majority of the patients (59.15%) were in the age group of 21-50 years.

Table 5.2: Distribution of cases in study group according to nature of procedure:

Type of procedure	No of cases	Percentage
Elective	34	34
Emergency	66	66
Total	100	100

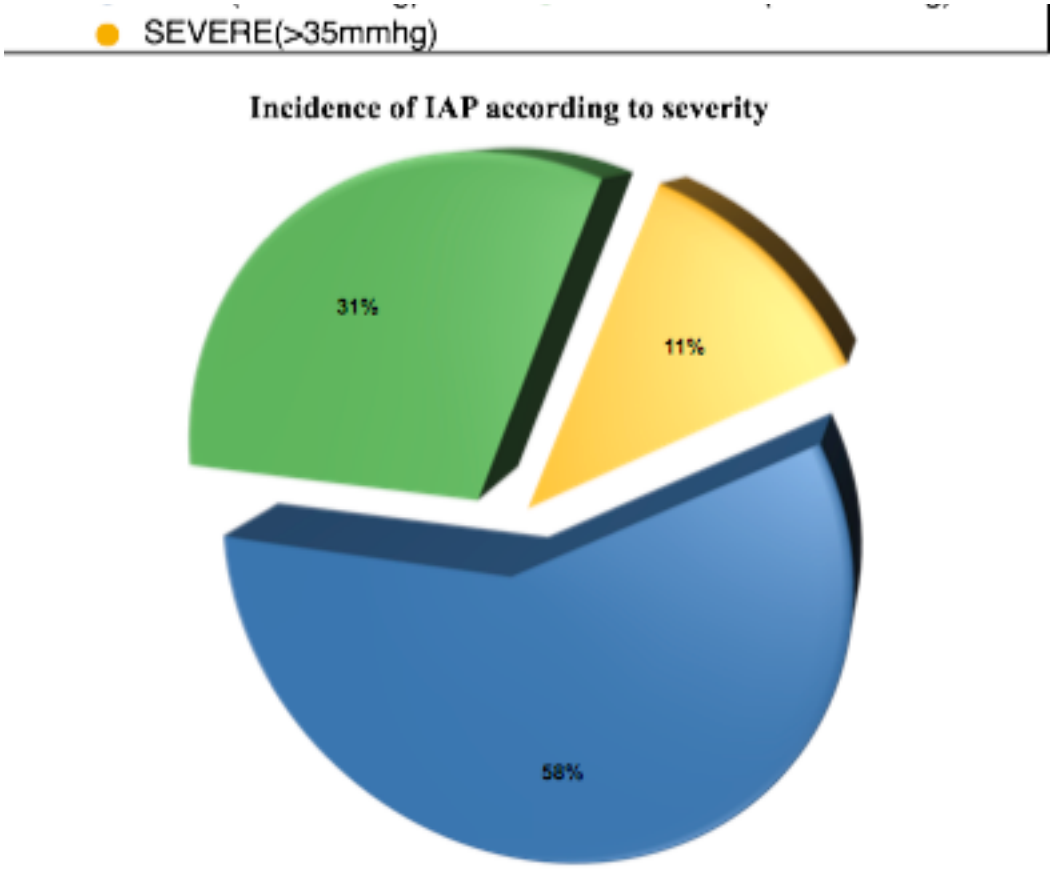
Graph5.2

66% of the cases were emergency procedures.

Table 5.3: Distribution of cases in study group according to Body Mass Index (BMI)

BMI	CASES	Percentage
< 18.5	7	7
18.5 – 25	89	89
25 – 30	4	4
Total	100	100

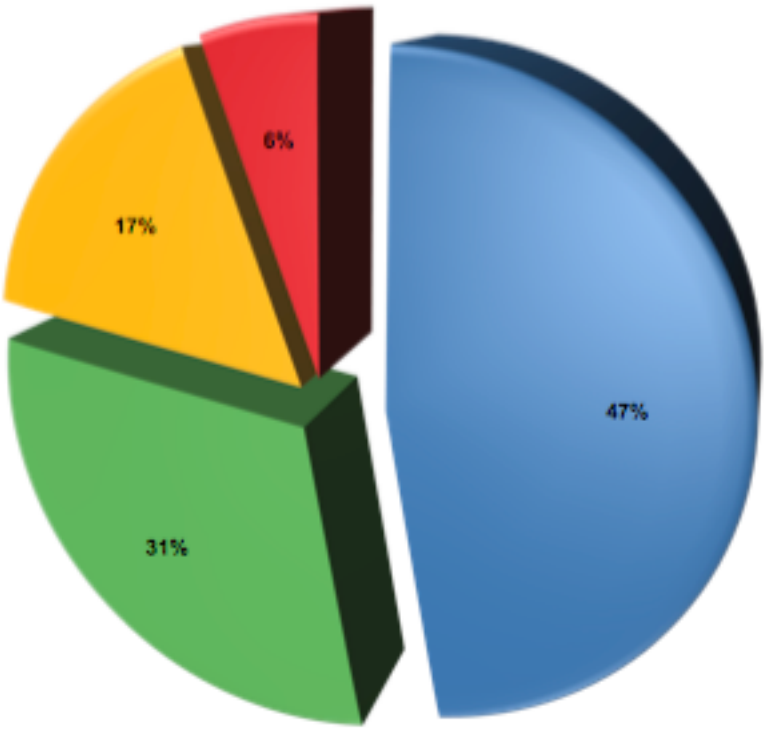
Graph 5.3



Majority (88.73%) of the patients were in the BMI group of 18.5 to 25.

Table 5.4: Incidence of Intra abdominal hypertension (IAH) according to severity:

Severity of Hypertension	No.of patients	Incidence(n=100)
Mild (10-20 mm of Hg)	21	21%
Moderate (21-35 mm of Hg)	11	11%
Severe (>35 mm of Hg)	4	4%
Total	36	36



The incidence of IAH in the current study was 36% with 21% having mild IAH, 11% having moderate IAH and 4% having severe IAH.

Table 5.5: Incidence of Intra abdominal hypertension according to Grade:

Grade	No.of patients
1	17
2	11
3	6
4	2
Total	36

Grade 1 IAH was seen in 17%, 11% had Grade 2 IAH, 5% had Grade 3 IAH and 2% had Grade 4 IAH.

Table 5.6: Correlation between IAP and BMI, Pulse, MAP, RR, A/G, U/O, SpO_2 from day 0 to day 7 in study group:

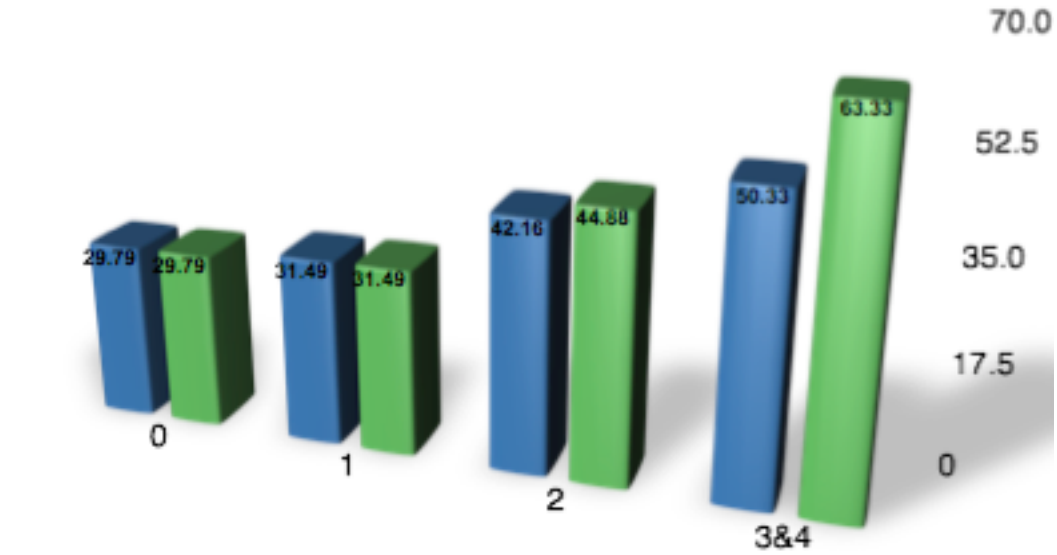
Correlation between IAP and		**Post operative**						
	Day 0	**Day 1**	**Day 2**	**Day 3**	**Day 4**	**Day 5**	**Day 6**	**Day 7**
BMI	0.14*	0.20*	0.17*	0.17*	0.17*	0.14*	0.07	0.02
PR	0.76#	0.88#	0.51#	0.43#	0.37#	0.35#	0.40#	0.34#
MAP	-0.76#	-0.83#	-0.39#	-0.34#	-0.28#	-0.33#	-0.35#	-0.36#
RR	0.83#	0.88#	0.67#	0.68#	0.55#	0.54#	0.61#	0.68#
U/O	-0.86#	-0.85#	-0.44#	-0.34#	-0.34#	-0.30#	-0.33#	-0.44#
A/G	0.45#	0.62#	0.30#	0.27#	0.25#	0.22$	0.19*	0.17*
SpO_2	-0.47#	-0.56#	0.02	-0.02	-0.05	-0.06	-0.09	-0.37#

*P<0.05 $P<0.005 #P<0.0001

There was significant association between PR, MAP, RR, U/O, A/G, SpO_2 and the IAP. However BMI did not show a high degree of association with IAP.

Table 5.7: Association of IAP with TLC on day 0 and maximum recorded TLC{TLC(max)}:

IAP Grade	N	TLC on day 0	TLC(max)
		Mean ± SD	Mean ± SD
0	64	9182 ± 1576	9182 ± 1576
1	17	9273 ± 2399	18676 ± 20494
2	11	12732 ± 1382	12924 ± 1760
3 & 4	8	13447 ± 2506	15513 ± 3369
F Value		48.83	12.96
P Value		<0.0001	<0.0001



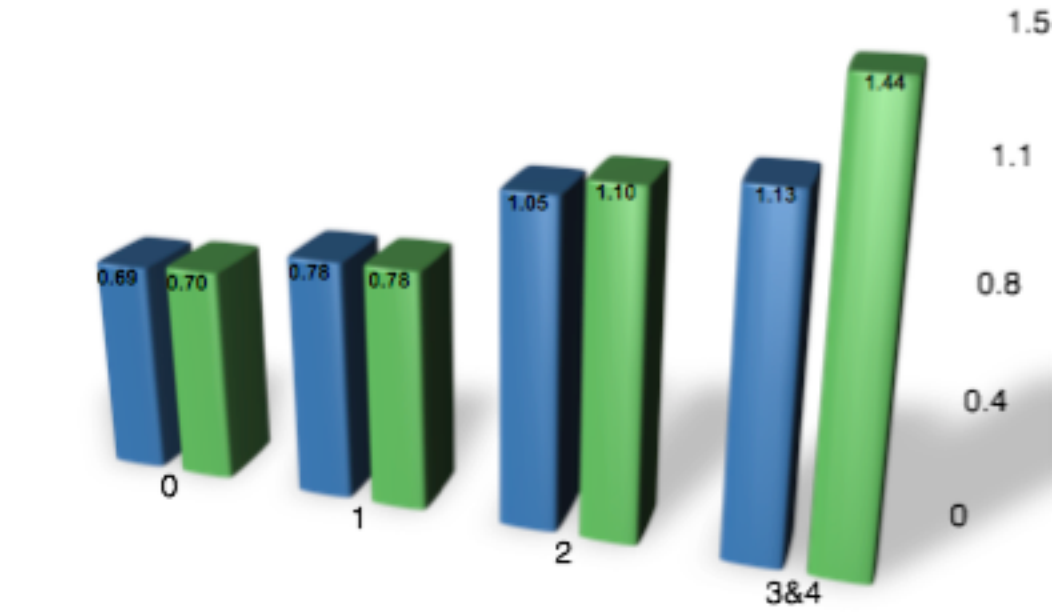
Graph 5.4:

There was statistically significant association between the IAP and TLC

Table 5.8: Association of IAP with BUN on day 0 and maximum recorded BUN {BUN(max)}:

IAP Grade	N	BUN on day 0	BUN(max)
		Mean ± SD	Mean ± SD
0	64	29.79 ± 4.37	29.79 ± 4.37
1	17	31.49 ± 3.31	31.49 ± 3.31
2	11	42.16 ± 5.81	44.88 ± 8.78
3 & 4	8	50.33 ± 10.18	63.33 ± 21.60
F Value		107.63	113.49
P Value		<0.0001	<0.0001

Graph 5.5

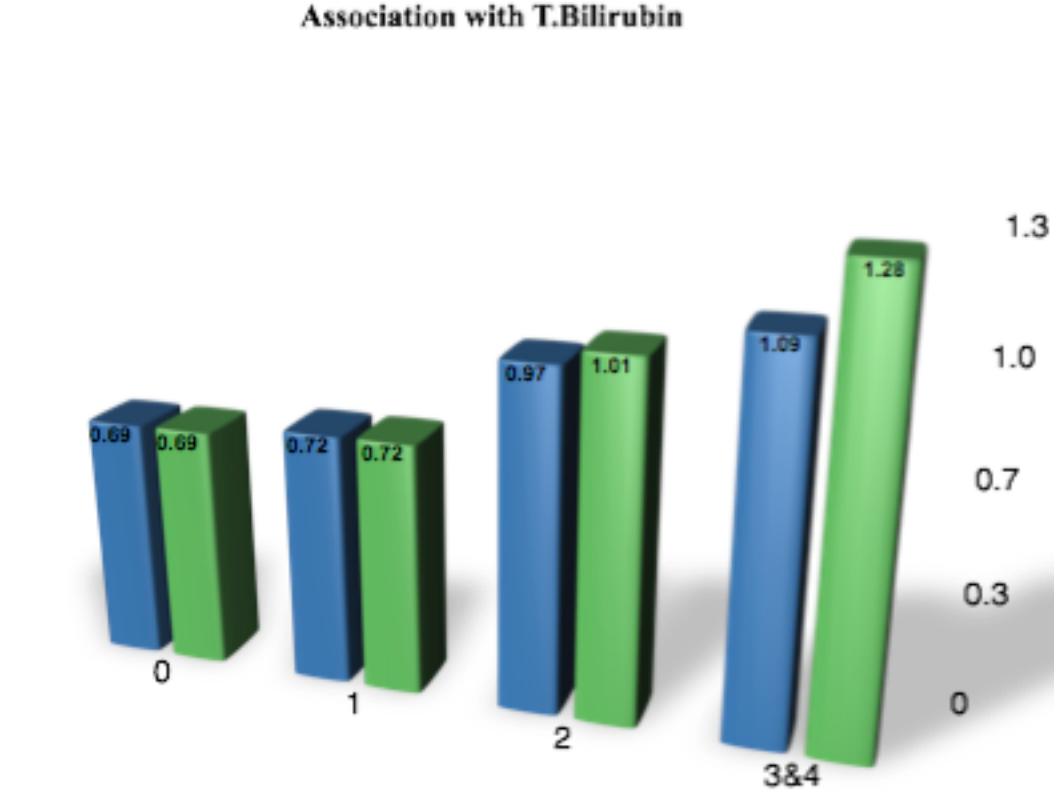


The F test and P value signify a high degree of association between the IAP and BUN levels.

Table 5.9: Association of IAP with Sr.Creatinine on day 0 and maximum recorded Sr.Creatinine {Sr.Creatinine(max)}:

IAP Grade	N	Sr. Creatinine on day 0	Sr. Creatinine (max)
		Mean ± SD	Mean ± SD
0	64	0.69 ± 0.13	0.70 ± 0.13
1	17	0.78 ± 0.13	0.78 ± 0.13
2	11	1.05 ± 0.14	1.10 ± 0.20
3 & 4	8	1.13 ± 0.31	1.44 ± 0.50
F Value		48.83	93.52
P Value		<0.0001	<0.0001

Graph5.6



The F test and P value signify a high degree of association between the IAP and Serum creatinine levels.

Table 5.10: Association of IAP with T.Bilirubin on day 0 and maximum recorded T.Bilirubin {T.Bilirubin(max)}:

IAP Grade	N	T. Bilirubin on day 0	T. Bilirubin (max)
		Mean ± SD	Mean ± SD
0	64	0.69 ± 0.17	0.69 ± 0.17
1	17	0.72 ± 0.22	0.72 ± 0.22
2	11	0.97 ± 0.16	1.01 ± 0.20
3 & 4	8	1.09 ± 0.24	1.28 ± 0.38
F Value		33.41	51.49
P Value		<0.0001	<0.0001

Graph 5.7

The F test and P value signify a high degree of association between the IAP and Total Bilirubin levels.

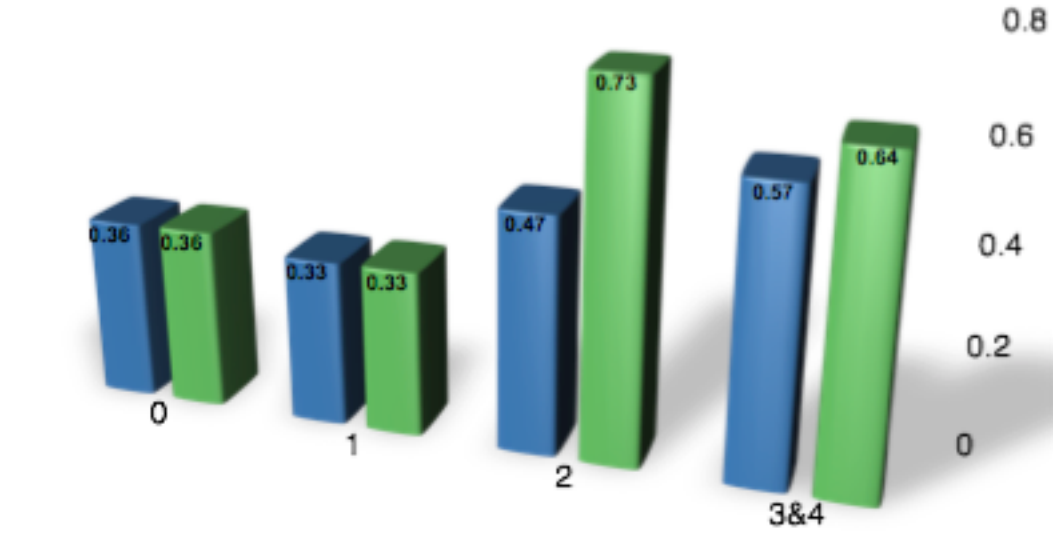


Table 5.11: Association of IAP with D.Bilirubin on day 0 and maximum recorded D.Bilirubin {D.Bilirubin(max)}:

IAP Grade	N	D. Bilirubin on day 0	D. Bilirubin (max)
		Mean ± SD	Mean ± SD
0	64	0.36 ± 0.08	0.36 ± 0.08
1	17	0.33 ± 0.12	0.33 ± 0.12
2	11	0.47 ± 0.08	0.73 ± 1.31
3 & 4	8	0.57 ± 0.19	0.64 ± 0.22
F Value		30.24	6.33
P Value		<0.0001	<0.0001

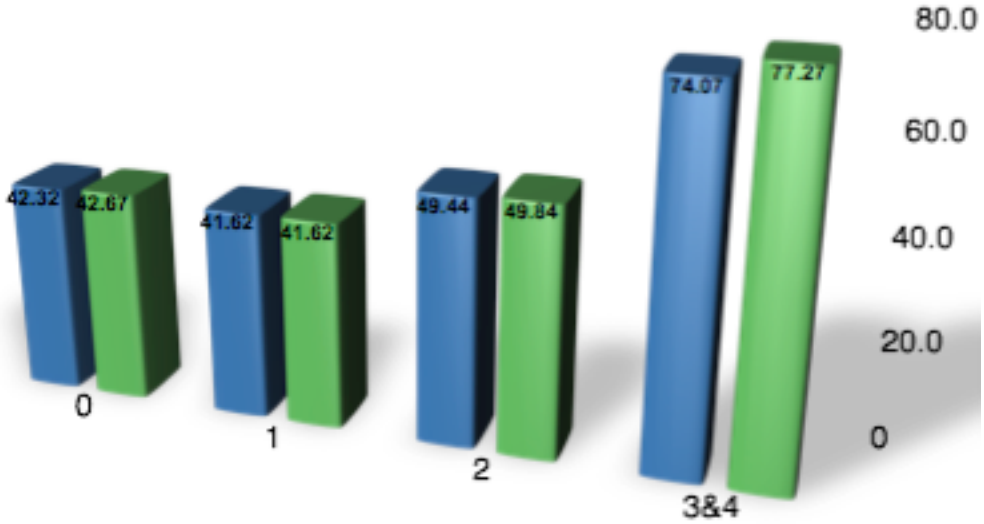
Graph 5.8

The F test and P value signify a high degree of association between the IAP and Direct Bilirubin levels.

Table 5.12: Association of IAP with ALT on day 0 and maximum recorded ALT {ALT(max)}:

IAP Grade	N	ALT on day 0	ALT(max)

Association with ALT



		Mean ± SD	Mean ± SD
0	64	42.32 ± 7.71	42.67 ± 7.69
1	17	41.62 ± 3.85	41.62 ± 3.85
2	11	49.44 ± 7.91	49.84 ± 8.18
3 & 4	8	74.07 ± 28.70	77.27 ± 27.98
F Value		46.94	57.98
P Value		<0.0001	<0.0001

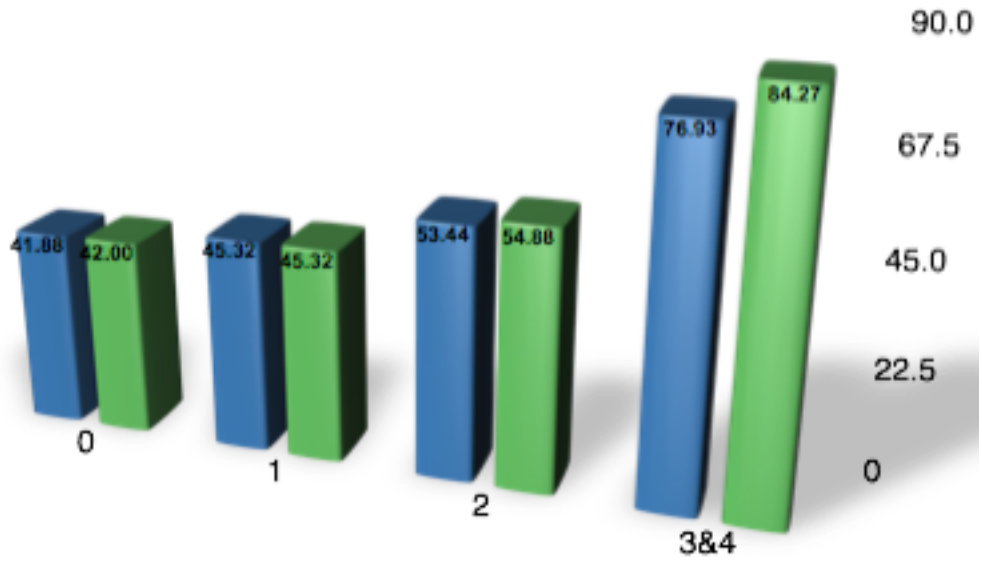
Graph 5.9

The F test and P value signify a high degree of association between the IAP and ALT levels.

Table 5.13: Association of IAP with ALP on day 0 and maximum recorded ALP {ALP(max)}:

IAP Grade	N	ALP on day 0	ALP(max)/day
		Mean ± SD	Mean ± SD
0	64	41.88 ± 8.06	42 ± 7.97
1	17	45.32 ± 6.94	45.32 ± 6.94

Association with ALP



2	11	53.44 ± 13.41	54.88 ± 12.87
3 & 4	8	76.93 ± 31.60	84.27 ± 33.58
F Value		48.83	59.95
P Value		<0.0001	<0.0001

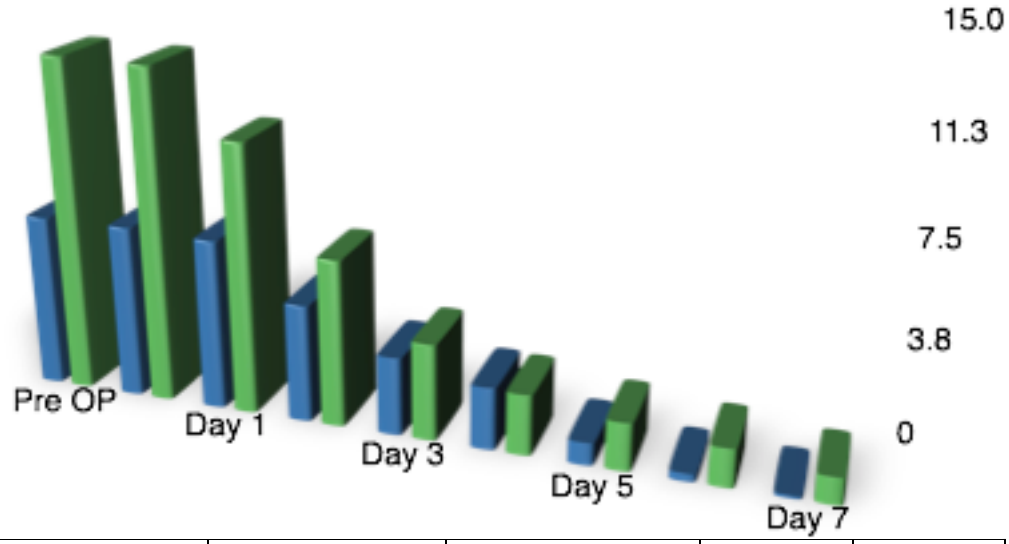
Graph 5.10

The F test and P value signify a high degree of association between the IAP and ALP levels.

Table 5.14: Comparison of Pre operative IAP and Post operative IAP in elective and emergency surgery group:

IAP on	**Elective**	**Emergency**	**Z Value**	**P Value**
	Mean ± SD (n=74)	**Mean ± SD (n=139)**		
Pre operative	6.70 ± 3.66	12.96 ± 8.25	7.64	<0.0001

Comparison on Pre and Post op IAP



Day 0	6.70 ± 3.66	12.86 ± 8.10	7.62	<0.0001
Day 1	6.59 ± 3.48	10.40 ± 9.80	4.11	<0.0001
Day 2	4.53 ± 3.70	6.42 ± 8.19	2.32	<0.05
Day 3	3.01 ± 3.54	3.71 ± 7.28	0.94	>0.05
Day 4	2.39 ± 4.88	2.32 ± 6.20	0.09	>0.05
Day 5	0.86 ± 3.54	1.83 ± 5.52	1.54	>0.05
Day 6	0.30 ± 1.77	1.46 ± 5.06	2.44	<0.05
Day 7	0.11 ± 0.93	1.02 ± 4.82	2.16	<0.05

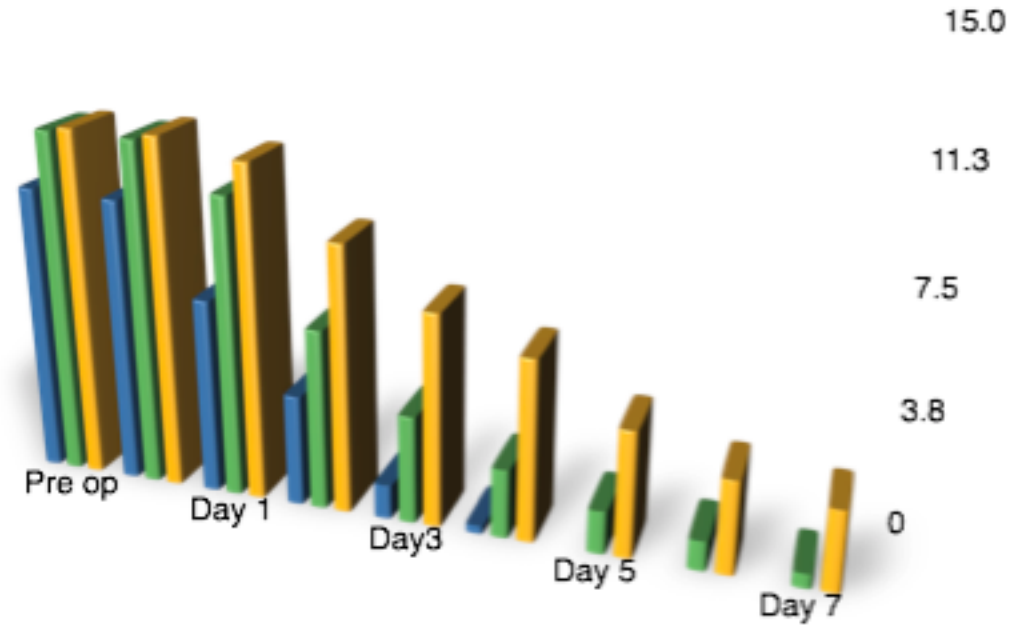
Graph 5.11

The mean IAP is significantly higher in the emergency surgery group as compared to the elective surgery group.

Table 5.15: Comparison of Pre Operative IAP and Post Operative IAP according to age:

IAP on	Age (≤ 20Yrs)	Age (21-50Yrs)	Age (>50Yrs)	F Value	P Value
	Mean ± SD	Mean ± SD	Mean ± SD		
Pre op	9.33 ± 3.25	11.21 ± 8.26	11.33 ± 9.34	1.23	>0.05
Day 0	9.22 ± 3.31	11.16 ± 8.05	11.33 ± 9.34	1.37	>0.05
Day 1	6.33 ± 3.39	9.70 ± 9.35	10.78 ± 8.95	3.94	<0.05
Day 2	3.59 ± 3.001	5.83 ± 7.22	8.61 ± 9.17	5.66	<0.005
Day 3	1.10 ± 2.09	3.47 ± 5.98	6.83 ± 9.06	9.65	<0.0001
Day 4	0.26 ± 0.79	2.21 ± 4.96	5.81 ± 9.69	10.78	<0.0001
Day 5	0 ± 0	1.37 ± 4.10	4.03 ± 8.82	7.55	<0.001
Day 6	0 ± 0	0.94 ± 3.29	2.97 ± 8.08	5.50	<0.005
Day 7	0 ±0	0.46 ± 2.47	2.56 ± 8.27	5.19	<0.01

Comparison of Pre and Post op IAP according to age



Graph 5.12

There was no significant association between age and the intra abdominal pressure.

Patients above the age of 50 years on an average required a longer time for the normalization of the IAP.

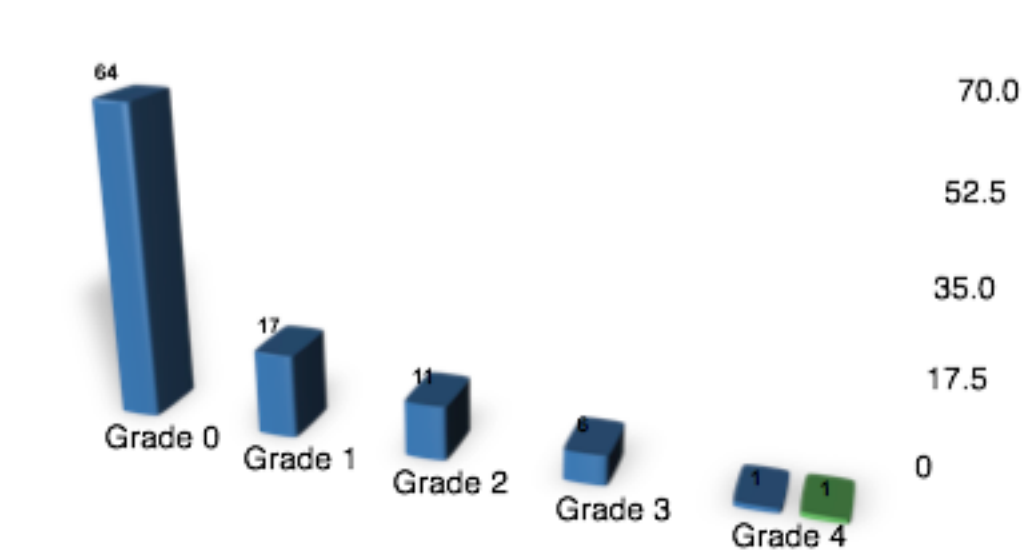
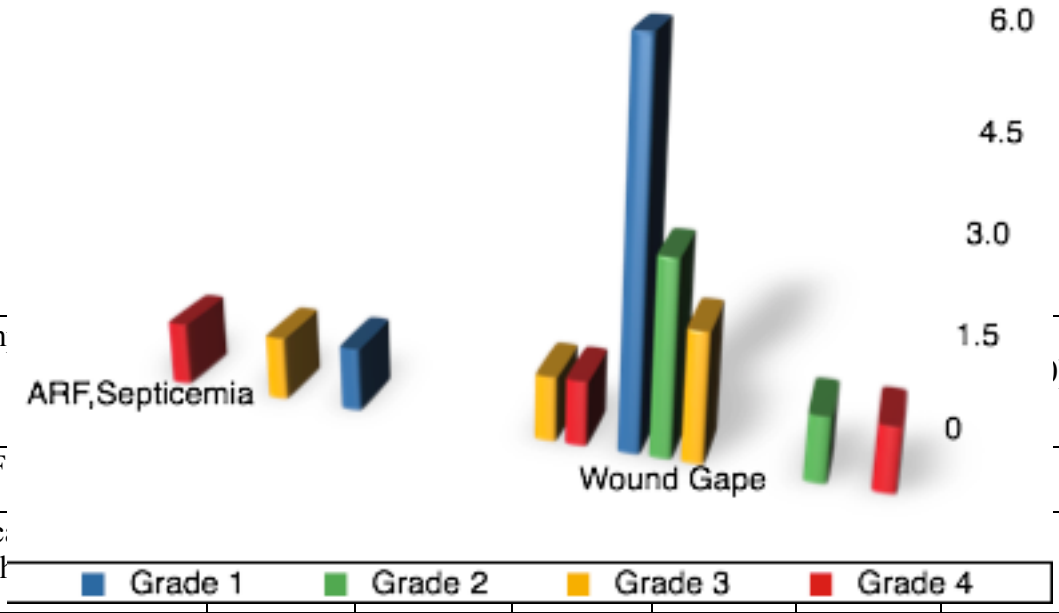


Table 5.16: Association between IAP grade and Outcome:

IAP grade	Outcome		Total
	Survived(%)	Death(%)	
0	64	0	64
1	17	0	17
2	11	0	11
3	6	0	6
4	1	1	2
Total	99	1	100

Graph 5.13

The single death in the study were associated with IAP of Grade IV which had a mortality rate of 50%.



Com						
ARF						
Faec empl						
Serous discharge from wound site	0	1	0	0	0	1
Wound dehiscence	0	0	0	1	1	2
Wound gape	2	6	3	2	0	13
Wound gape, oliguria	0	0	1	0	1	2
Total	2 (2)	7 (40.54)	4(24)	4 (66.67)	8 (88.89)	25(17.84)

Table 5.17: Association between IAP grade and complications in study group

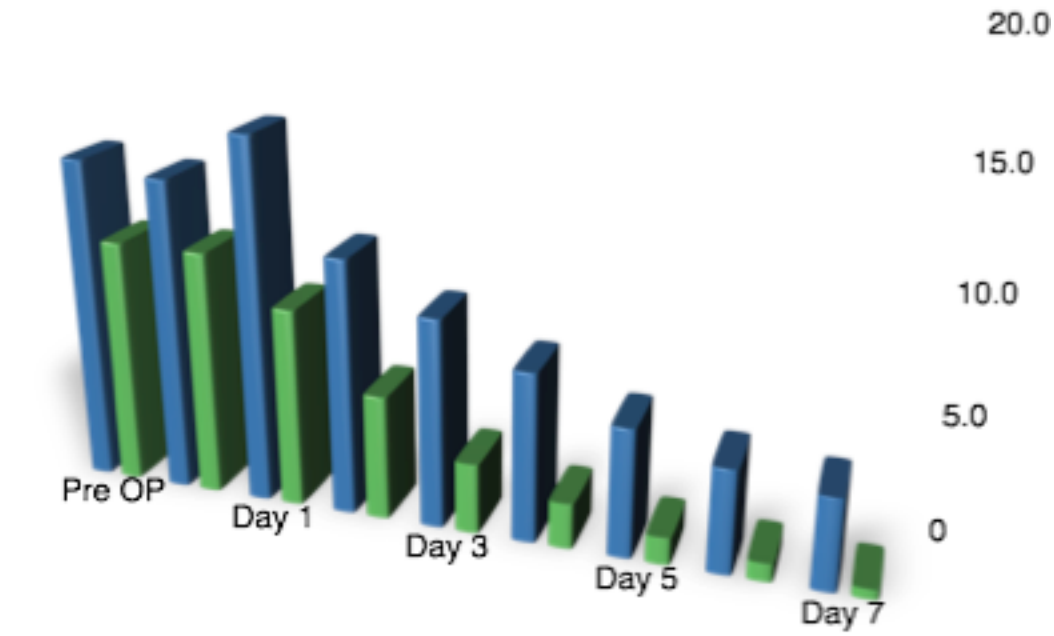
Graph 5.14

There was a very high complication rate of 66.67% and 88.89% associated with Grade III and IV of IAP respectively.

Table 5.18: Comparison of IAP in cases with and without Diabetes Mellitus (DM):

IAP on	DM Present	DM Absent	Z Value	P Value

	Mean ± SD (n=17)	**Mean ± SD (n=83)**		
Pre operative	13.9 ± 13.1	10.52 ± 6.91	1.04	>0.05
Day 0	13.4 ± 12.1	10.49 ± 6.93	0.98	>0.05
Day 1	15.5 ± 13.7	8.52 ± 7.54	2.07	>0.05
Day 2	10.9 ± 12.1	5.32 ± 6.25	1.88	>0.05
Day 3	8.9 ± 10.8	3 ± 5.47	2.22	<0.05
Day 4	7.2 ± 10.3	1.93 ± 5.02	2.08	>0.05
Day 5	5.47 ± 9.82	1.15 ± 4.14	1.80	>0.05
Day 6	4.41 ± 9.83	0.77 ± 3.26	1.52	>0.05
Day 7	3.9 ± 10.5	0.43 ± 2.63	1.35	>0.05



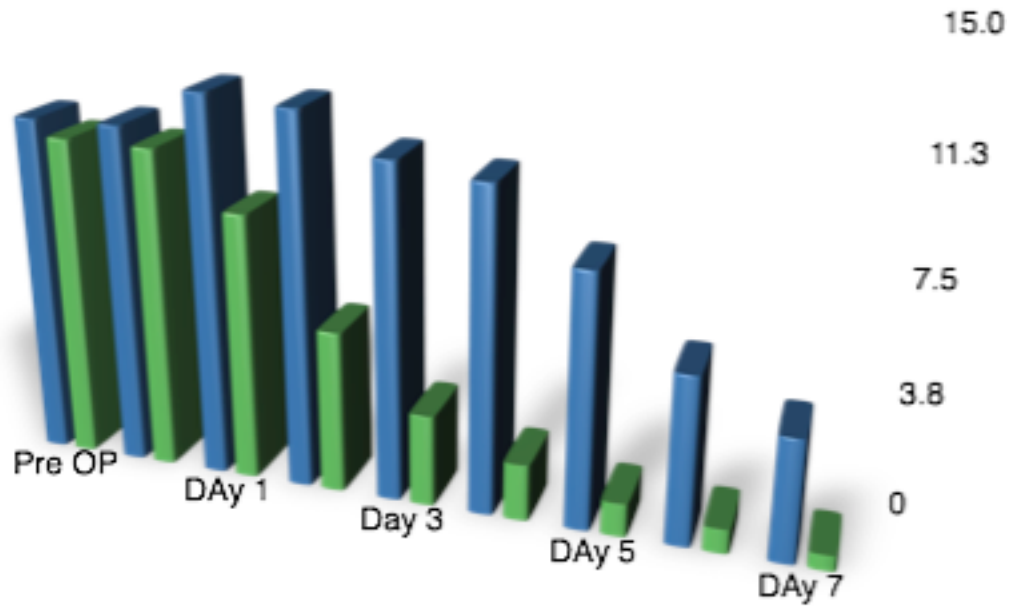
Graph 5.15

There was no statistically significant association between IAP and Diabetes Mellitus.

Table 5.19: Comparison of IAP in cases with and without Hypertension (HTN):

IAP on	HTN Present	HTN Absent	Z Value	P Value
	Mean ± SD (n=12)	Mean ± SD (n=88)		
Pre operative	11.33 ± 7.98	10.75 ± 7.60	0.25	>0.05
Day 0	11.33 ± 7.98	10.69 ± 7.47	0.27	>0.05
Day 1	12.58 ± 9.61	8.87 ± 8.27	1.31	>0.05
Day 2	12.3 ± 11.1	5.37 ± 6.54	2.16	>0.05
Day 3	11 ± 11.9	3.02 ± 5.47	2.32	<0.05
Day 4	10.6 ± 13.7	1.86 ± 4.53	2.19	>0.05
Day 5	8.3 ± 11	1.09 ± 4.04	2.25	<0.05
Day 6	5.50 ± 8.58	0.79 ± 3.72	1.89	>0.05
Day 7	4 ± 7.24	0.51 ± 3.61	1.66	>0.05

Association of SHT with IAP in Pre and Post op patients



Graph 5.16

There was no statistically significant association between IAP and systemic hypertension.

6.Discussion

Discussion

A compartment syndrome is a condition in which increased pressure in a confined anatomical space adversely affects the function and viability of the tissues therein. Confined anatomical spaces mostly associated with compartment syndromes are the fascial spaces of the extremities, the orbital globe (glaucoma), the cranial cavity (epidural/subdural hematoma), the kidney capsule (post-ischemic oliguria) and the abdominal cavity.

ACS is a condition in which sustained increased pressure within the abdominal wall, pelvis, diaphragm, and the retroperitoneum, adversely affects the function of the entire gastrointestinal tract and connected extraperitoneal organs. It usually requires operative decompression.

IAH is graded as [33]:

- Mild IAH

  10-20 mm Hg: Clinically not significant changes occur which usually doesn't need surgical intervention

- Moderate IAH

  21-35 mm Hg: There may be certain critical changes occurring and might need interventions

- Severe IAH

>35 mm Hg: Abdominal compartment syndrome- needs definitive surgical intervention.

Five organs of the abdomen were subjective to volume change and pressure[37].

1) Solid organs - Liver, spleen causes chronic abdominal hypertension.

2) Hollow viscus leads to inflammation , ileum or bowl obstruction.

3)Fluid overload in a patient the blood and lymphatics causes abdominal hypertension.

4) the peritoneum usually absorbs large amount of fluid incase of inflamation

5) peritoneal cleft also accumulates a large amount of fluid

It is yet to prove in clinical settings which of the above five causes is the reason for the increase in the volume which is the imajor cause of IAH.

The peritoneum covers 1.8 m^2 of the body surface. It covers the whole of intestinal organs.On inflammation only 0.5cm increase in thickness noted and there will be absorption of 1.8 m^2 = 18,000 cm^2 x 0.5 cm thickening = 9,000 ml of fluid due to inflammation of peritoneum. Hence fluid shift will be seen in burns.

Due to its large surface area there will be large amounts of transudates and exudates formed in a short time due to irritation or injury.

Abdominal wall compliance plays an important role in the regulation of the intra abdominal pressure:

The dynamic relation between volume and pressure within the abdomen is important because after a relatively long period of compensation, deterioration is fast due to limited abdominal wall compliance. Compliance is structurally dependent on the stiffness of the peritoneum and its volume-pressure curve (i.e. compliance is not linear).

Upon IAP increase, abdominal wall fasciae stretch and lose expandability. Progressively smaller volume increments are required to further elevate IAP[28]. Conversely, high IAP may be dramatically relieved by decompression.

Due to abdominal hypertension- shallow respiration high diaphragm on percussion, low output and increased central venous pressure[44]. Multi organ dysfunction progresses unless IAP is reduced. Hence the requirement of timely intervention either fluid management or surgical decompression reverses the effects.

Radiologically computed tomography yields increases in anteroom-posterior to transverse diameter, renal compression, bowel thickening and inguinal herniation[45].

Age:

The mean age in our study was 34.48 years. In a study by Khan S et al. the mean age was 34.78 years[46]. Cheatham *et al*. have reported a mean age of 51±19 years, Meldrum *et al*. 39±9 years, and Hong *et al*. 42 years[47-49].

In the current study there was no statistically significant association between age and IAP **(Table 5.1; Graph 5.1)**. However the normalization of IAP took a longer time in the patients above 50 years of age. **(Table 5.15; Graph 5.12)**

Sex:

There were 141 males and 72 females (66.2% males and 33.8% females) in the current study. A similar ratio was seen in the studies by Khan S et al. (76% males), Hong *et al*. (72% males), Meldrum *et al*. (70% males), Sugrue *et al*. and Cheatham *et al*. (60% males)[46-50]. There was no significant correlation between sex and IAP in the current study **(Table 5.1; Graph 5.1)**.

Incidence:

The incidence of IAH and ACS reported by various studies ranges from 2 to 78% and 0.5 to 36%, respectively, and depends on the population and the values used to define these entities[49].

In the current study the incidence of IAH was 36.15% and that of ACS was 4.69% **(Table 5.4, 5.5)**.

The lower incidence observed was because this study includes low-risk (cholecystectomy, appendectomy etc) as well as high-risk patients (peritonitis, intestinal obstruction, perforation etc) whereas most of the previous studies confined data collection to high-risk patients. While the earlier approach ensures a good yield of patients with ACS, it may result in a very high incidence compared with that seen clinically in the general

population overall. Furthermore, such an approach potentially misses those patients who are not at high risk, and yet may

have Multiple Organ Dysfunction Syndrome (MODS) falsely attributed to sepsis or irreversible shock when in fact they have unrecognized ACS. By measuring the IAP prospectively in all patients, this study obtained true overall incidence.

A similar study by Khan S et al. revealed an incidence of 80% for IAH and 3.05% for ACS[46].

Changes in physiology that are seen with increasing pressure involve almost all systems and overlap.

Cardiovascular system:

Adverse effects with IAP's will occur even when the pressure is as low as 10-15 mm Hg.

Increased IAP's causes fall in cardiac output blood pressure remains unaltered. Tachycardia occurs in order to maintain cardiac output.

Increase in vascular resistance may be due to mechanical compression of capillary or nitric acid deficiency.

In a study by Chang MC et al, intra abdominal hypertension of >25 mm of Hg led to a significant increase in heart rate of up to 124±18 /min(51).

In an another study by Lazaro Gotloib et al heart rate showed significant increase with intra abdominal pressures of more than 15 cm of H_2O[52].

The results of the current study were among similar lines ($r=0.34$ to 0.88; $p<0.0001$) **(Table 5.6)**.

In the study by Chang MC et al, intra abdominal hypertension led to a fall in the mean arterial pressure[51].

In a study of 46 patients by Widergreen et al, the mean arterial pressure at 40 cm of H_2O bladder pressure was 86 mm of Hg which increased to 92 mm of Hg when the bladder pressure reduced to 22 cm of H_2O[53].

In the study by Lazaro Gotloib et al, mean arterial blood pressure showed significant increase with intra abdominal pressures of more than 10 cm of H_2O[52].

In the current study there was a significant negative correlation between MAP and IAP (-0.76 to -0.28; $p<0.0001$) **(Table 5.6)**.

According to Malbrain et al Intra abdominal hypertension has a statistically significant association with acidosis[38]. In the current study, out of the 10 patients who underwent Arterial blood gas analysis(ABG) 8 cases showed acidosis and all of them were associated with mortality. In the current study ABG was performed only in those cases where deemed necessary and as such statistical correlation was not possible. This is one of the limitations of our study.

Total Leucocyte Count (TLC):

In a study of a total of 75 patients by Cem Ibis and Aydin Altan there was a statistically significant correlation between IAP and TLC ($p=0.002$)[40].They concluded that the determination of the

WBC count only is not safe enough to diagnose acute abdomen. The interpretation of BP level together with WBC count seems to be more effective because of the statistically significant difference between the test and the control group related to the elevated WBC count.

Similar results were obtained in our study which showed a highly significant association between the IAP and TLC ($p<0.0001$) **(Table 5.7; Graph5.4)**.

Respiratory function:

Increase in IAP and decrease thoracic volume pushes the diaphragm up. Atelectases of pleural cavity and decrease in alveolar clearance occur due to decreased volume. Early complication of abdominal hypertension due to peritonitis is Pneumonia.

However there were no cases of pneumonia in the current study.

Raise in thoracic pressure, low cardiac output and pulmonary vascular resistance occur when there is diaphragmatic protrusion into pleural cavity. Hypoxemia, hypercarbia and acidosis occur due to ventilation/ perfusion abnormality. When the pressure is 20mm Hg physiological dysfunction occurs.

As noted by David Hopkins and S.W. Gemmell [54] there was a significant increase in the respiratory rate even in the current study ($r=0.54$ to 0.88; $p<0.0001$) **(Table 5.6)**.

In the current study there was a significant association between

SpO_2 and intra abdominal pressure ($P<0.0001$) **(Table 5.6)** which concurs with results of the available literature. However all the modern day studies measured the association of the IAP and respiratory function by various scientifically advanced parameters like PaO_2 (partial pressure of oxygen), FiO2 (fraction of inspired oxygen), QI/Qt (intrapulmonary shunt fraction) , PIP (peak inspiratory pressure), PEEP (positive end-expiratory pressure) and Cdyn (dynamic compliance) which were not used in our study and is one of the limitations of our study.

Renal function:

Complications such as oliguria; anuria usually ensues with higher pressures.

Mainly due to the decrease in renal blood flow, GFR, urine output, and various specific tubular functions associated with raised IAP is of multifactorial etiology.

Improved cardiac output plays a role in diminished renal perfusion but even when cardiac output is maintained at normal or supernormal values by blood volume expansion, impairment of renal function persists. Renal dysfunction is also caused by compression of the renal vein, which causes partial renal blood

outflow obstruction. Compression of the abdominal aorta and renal arteries contributes to increased renal vascular resistance. Furthermore, direct compression of the kidneys elevates cortical pressures, leading to a "renal compartment syndrome". Elevation of plasma anti diuretic hormone may represent another etiological factor.

Savino JA et al in a study of 51 patients noted a mean urine output of 47 ml/hr at a mean abdominal pressure of 33.5 cm of H_2O which increased to 55ml/hr after the mean abdominal pressure reduced to 19.1 cm of H_2O[55].

In a study of 46 patients Widergreen et al, a mean urine output of 79ml/hr was noted at a bladder pressure of 40 cm of H_2O which increased to 123ml/hr after the mean abdominal pressure reduced to 22 cm of H_2O[53].

In the same study there was also a significant association between the intra abdominal pressure and Blood urea and serum creatinine levels[53].

In the current study there was a significant negative correlation between urine output and IAP ($r = -0.3$ to -0.86; $p<0.0001$). The current study also revealed a significant association of IAP with Blood urea and serum creatinine levels **(Table 5.6, 5.8, 5.9; Graph 5.5, 5.6)**.

46.67% of patients with ACS developed oliguria and 33.3% of patients with ACS progressed to ARF and anuria in the current study **(Table 5.17; Graph 5.14)**.

Effects on liver function:

Increase in IAP affects the hepatic arteries and the portal blood flow. Trauma patients are more susceptible because of shock induced intestinal vascular resistance.

Synthesis of hepatic acute-phase protein, immunoglobulin and host defense system were impaired due to decrease in hepatic blood flow.

Detailed studies addressing the issue of reduced hepatic protein synthesis have not yet been published.

Transient alterations of hepatic enzymes are frequently observed after uneventful laparoscopic cholecystectomy, presumably attributed to the elevated intraabdominal pressure of the pneumoperitoneum according to Marakis et al[(62)].

Aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, and total bilirubin levels were significantly elevated following ACS and sepsis in a study performed in rats by Tolga MA et al[(57)].

The current study reveals a significant association between IAP and the four liver function parameters assessed i.e., Total bilirubin, direct bilirubin, serum ALP and serum ALT **(Table 5.10, 5.11, 5.12, 5.13; Graph 5.7, 5.8, 5.9, 5.10)**.

Wound healing:

Increase in IAP impairs the wound healing due to reduced abdominal wall and fascia blood flow. Abdominal binder must be avoided as it further reduces the blood flow.

Rectus sheath blood flow was significantly reduced at all pressure levels when compared to baseline and negatively correlated (r = -0.82) with increasing IAP in a porcine model by Diebel L et al[58].

31.17% of patients with IAH developed wound gape and 13.3% of patients with ACS developed wound dehiscence **(Table 5.17; Graph 5.14)** (Figure 5.1,5.2).

Gastrointestinal function:

When IAP is 15mmHg, decrease in splanchnic circulation occurs. As it is documented many of the abdominal arteries, veins and lymphatics have low perfusion. Alterations of pH, bacterial growth translocation, motility disorder, harmone abnormality and exocrine dysfunction were also documented as secondary effects. Effects on spleen, pancreas, adrenal and reproductive organs are yet to be documented.

Impairment of arterial flow - Abdominal hypertension impairs intestinal blood flow. Elevation in IAP results in decreased mesenteric arterial blood flow; intestinal mucosal blood flow; and arterial perfusion of the stomach, duodenum, intestine, pancreas, and spleen. As IAP increases, mucosal pH falls, indicating severe ischemia or necrotizing pancreatitis.

Compartment-induced impaired intestinal perfusion may be a critical factor in anastomotic healing. Abdominal hypertension probably plays a role in many of the organ dysfunctions of currently questionable etiology. Examples may be ischemic gastritis, acalculous cholecystitis or pancreatitis, colon ischemia, and some forms of bowel ischemia.

These changes are greater than can be accounted for by the alterations in cardiac output and also occur when cardiac output and systemic blood pressure are maintained at normal levels.

Effects on abdominal veins

Even a mild elevation of IAP in cirrhotic patients cause increase in hepatic venous pressure and azygos blood flow increases. Vice versa occurs once IAP is lowered. It’s a controversy whether increase in IAP precipitates variceal bleed.

Effects on lymph flow

Increase in IAP significantly reduce lymphatic flow of thoracic duct. the same increases after decompression. Transfer of

peritoneal fluid into the thoracic lymphatics decreases due to stretching of diaphragm.

Translocation:

Due to decreased intestinal perfusion caused by increased IAP, traslocation of bacteria occurs in high rates. This causes increase in infection rate and sepsis which may lead to further septic complications.

According to Diebel L et.al, increased IAP of 25 mm of Hg leads to decreased mucosal blood flow and to bacterial translocation, which may contribute to later septic complications and organ failure[58].

In the current study 33.3% of patients with ACS developed septicaemia **(Table 5.17;Graph 5.14)**.

Intracranial pressure (ICP):

Idiopathic intracranial hypertension is accentuated due to chronic abdominal hypertension. During lapraoscopy abdominal pressure increases which causes increase in intracranial pressure.

Nature of surgery: (Emergency Vs Elective Surgery):

In a large prospective study from 1999, Sugrue et al. studied IAH in relation to renal impairment in 263 patients admitted to the ICU after emergent (n = 174) or elective (n = 89) abdominal surgery IAP 18 mmHg or greater was found in 41%.

IAH after emergency surgery was seen in 46% of patients after upper and 46 per cent after lower gastrointestinal surgery compared with 60 per cent after vascular surgery. The corresponding findings after elective surgery were 29%, 12%, and 32%, respectively, emphasizing the increased incidence of IAH/ACS after emergency surgery[59].

They also found the incidence of IAH in postoperative ICU patients after elective upper gastrointestinal surgery to be 29 per cent. This difference might be attributable to the higher IAH incidence in ICU patients in general[59].

Scollay et al. studied IAP prospectively in 42 patients recovering from elective major upper gastrointestinal surgery and found that 12 per cent had a transient IAH without an impact on postoperative organ function. No patient developed ACS and there were no deaths. The only significant finding was delayed return to oral diet in patients with transient IAH[60].

In the current study in emergency surgeries (n=139), the pre operative IAP (Mean ± SD) was 12.96 ± 8.25 cm of H_2O and in elective surgeries (n=74), the pre operative IAP (Mean ± SD) was 6.70 ± 3.66 cm of H_2O and the association was statistically very significant (p value = <0.0001) **(Table 5.14; Graph 5.11)**.

Mortality:

Cheatham *et al*. had found that elevated IAP alone does not have sufficient sensitivity or specificity to be useful as a predictor of mortality[47].

In a study by Hong JJ et al, 50% of the patients with ACS died as did 22.2% of patients with IAH[56].

A mortality rate of 100% was seen in ACS group and 13.2% in IAH group in a study by Khan S et al when decompression was not done, supporting the view that ACS, if left untreated, is invariably fatal[46].

In the current study a mortality rate of 33.3% was seen in ACS group and 6.49% in IAH group **(Table 5.16;Graph 5.13)**.

Hence, future studies on this subject should aim at devising a protocol which may help the healthcare professionals in early identification of the IAH and ACS patients and thus minimize the resulting high mortality.

Association with co-morbidities [Diabetes mellitus (DM), Hypertension (HTN) and Body Mass Index(BMI)]:

Although the association of Chronic IAH with co-morbidities is well documented, literature correlating IAH and co-morbidities is scanty.

Varela JE et al performed a study to examine the correlation between the IAP and obesity-related co-morbidities. Systemic hypertension was significantly associated with an elevated IAP but there was no significant association with diabetes mellitus[61].

In the current study there was no statistically significant association between IAP and DM and HTN ($p>0.05$) **(Table 5.18, 5.19; Graph 5.15,5.16)**.

Varela JE et al described a significant correlation between IAP and BMI[59].

According to Wilson et.al, elevated BMI does impact IAP, but the incremental value is small. Markedly increased IAP should not be attributed solely to elevated BMI and should be recognized as a pathologic condition[62].

There was a similar statistically significant correlation between the BMI and IAP even in the current study **(Table 5.2, 5.6; Graph 5.2)**.

6.Conclusion

Conclusions:

1. The incidence of IAH in the current study was 36%.
2. There was significant association between Pulse rate, Mean arterial pressure, Respiratory rate, Urine output, Body mass index, Abdominal girth, Oxygen saturation and the Intra abdominal pressure.
3. There was significant association between Total leucocyte count, Liver function tests, Renal function tests and intra abdominal pressure.

4. Patients with Grade III and IV Intra abdominal hypertension were associated with a higher rate of mortality and morbidity.
5. There was no significant association between intra abdominal pressure and co-morbidities like Diabetes Mellitus and Hypertension.
6. Incidence of intra abdominal hypertension was higher in emergency laparotomies as compared to elective laparotomies.

Summary:

Introduction: Abdominal compartment syndrome is one which pressure increases in a confined anatomical space and affects its function and viability of the tissue. In abdominal hypertension there is increase in volume in its contents IAP impair physiology and organ function, because of the limited compliance of abdominal wall. This study is being undertaken to evaluate the impact of IAP on outcome in patients undergoing laparotomies.

Materials and Methods: The detailed case history of two hundred and thirteen cases was recorded, clinical examination and investigations carried out. Patients undergoing elective and emergency laparotomies were allotted under Group A and Group B respectively. Intra-Abdominal pressure was monitored daily till the IAP normalized or till post operative day 7 along with the pulse, blood pressure, respiratory rate, oxygen saturation, abdominal girth, urine output and arterial blood gas in patients who underwent a laparotomy. All these factors were used to monitor the progress and assess the recovery of the patient.

Observation and Results: Of the 100 cases in the study, 66.2% of the patients were male and majority of the patients (59.15%) were in the age group of 21-50 years. 65.26% of the cases were emergency procedures. The incidence of IAH in the current study was 36.16% with 20.66% having mild IAH, 11.27% having moderate IAH and 4.23% having severe IAH. There was significant association between PR, MAP, RR, U/O, A/G, SpO_2 and the IAP. However BMI did not show a high degree of association with IAP. There was also significant association between the IAP and Liver and

Renal function tests. The mean IAP is significantly higher in the emergency surgery group as compared to the elective surgery group. There was a very high complication rate of 66.67% and 88.89% associated with Grade III and IV of IAP respectively. The single death in the study were associated with IAP of Grade IV which had a mortality rate of 50%.

Conclusions: There was significant association between Pulse rate, Mean arterial pressure, Respiratory rate, Urine output, Body mass index, Abdominal girth, Oxygen saturation and the Intra abdominal pressure. There was significant association between Total leucocyte count, Liver function tests, Renal function tests and intra abdominal pressure.

Patients with Grade III and IV Intra abdominal hypertension were associated with a higher rate of morbidity and mortality. There was no significant association between intra abdominal pressure and co-morbidities like Diabetes Mellitus and Hypertension. Incidence of intra abdominal hypertension was higher in emergency laparotomies as compared to elective laparotomies.

7.References

References:

1. Barnes GE, Laine GA, Giam PY, Smith EE, Granger HJ. Cardiovascular responses to elevation of intra-abdominal hydrostatic pressure. Am J Physiol. 1985;248:R209-13.

2. Mohapatra B. Abdominal compartment syndrome. *Indian J Crit Care Med*2004;8:26-32.

3. Rotondo MF, Cheatham ML, Moore FA, Reilly PM. Abdominal Compartment Syndrome-Symposium. *Contemporary Surgery*2003:59:260-70.

4. Bailey J, Shapiro MJ. Abdominal Compartment Syndrome. *Crit Care*2000;11:156-71.

5. Emerson H. Intra-abdominal pressures. *Arch Intern Med*1911;7:754-84.

6. Moore AFK, Hargest R, Martin M, Delicata RJ. Intra-abdominal hypertension and the abdominal compartment syndrome. *Br. J Surg*2004;91:1102-10.

7. Thorington JM, Schmidt CF. A study of urinary output and blood-pressure changes resulting in experimental ascites. Am J Med Sci 1923;165:880-90.

8. Bellis CJ, Wangensteen OH. Venous circulatory changes in the abdomen and lower extremities attending abdominal distention. Proc Soc Exp Biol Med 1939;4:490-8.

9. Ogilvie WH. The late complications of abdominal war wounds. Lancet 1940;2:253-6.

10. Bradely SE, Bradely GP. The effect of increased intra-abdominal pressure on renal function in man. J Clin Invest 1947;26:1010-15.

11. Gross RE. A new method for surgical treatment of large omphaloceles. Surgery 1948;24:277-92.

12. Baggot MG. Abdominal blow-out: a concept. Current Research Anesthesia Analgesia 1951;30:295-8.

13. Gordon ME. The acute effects of abdominal paracentesis in Laennec's cirrhosis upon changes of electrolytes and eater, renal function and hemodynamics. Am J Gastroenterol 1960;33:15-37.

14. Suazzi M, Polese A, Magrini F, et al. Negative influence of ascites in the cardiac function of cirrhotic patients, Am J Med 1975;59:165-70.

15. Cruikshank DP, Buschsbalm HJ. Effects of rapid paracentesis, cardiovascular dynamics and body fluid composition. JAMA 1973;225:1361-2.

16. Knauer CM, Love HM. Hemodynamics in cirrhotic patient during paracentesis. N Engl J Med 1967;276:491-6.

17. Sönderberg G, Westin B. Transmission of rapid pressure increase from the peritoneal cavity to the bladder. Scan J Urol Nephrol 1970;4:155-65.

18. Shenansky JH, Gillenwater JY. The renal hemodynamic and functional effects of external counterpressure. Surg Gynecol Obstet 1972;134:253-8.

19. Ivankovich AS, Albrecht RF, Zahed B et al. Cardiovascular collapse during gynecological laparoscopy. Ill Med J 1974;145:58-61.

20. Motev M, Ivankovich AD, Bieniarz J et al. Cardiovascular effects and acid base and blood gas changes during laparoscopy. Amer J Obstet Gynecol 1973;116:1002-1012.

21. Lenz RJ, Thomas TA, Wilkins DG. Cardiovascular changes during laparoscopy. Anaesthesia 1976;31:4-12.

22. Richardson JD, Trinkle JK. Hemodynamic and respiratory alterations with increased intra-abdominal pressure. J Surg Res 1976;20:401-4.

23. Kashtan J, Green JF, Parson EQ et al. hemodynamic effects of increased abdominal pressure. J Surg Res 1981;30:249-55.

24. Harman PK, Kron IL, McLachan DH et al. Elevated intra-abdomial pressure and renal function. Ann Surg 1982;196:594-7.

25. Richards WO, Scovill W, Shin B et al. Acute renal failire associated with increased intra-abdominal pressure. Ann Surg 1983;197:183-7.

26. Le Roith D, Bark H, Nyska M et al. The effect of abdominal pressure on plasma antidiuretic hormone levels. J Surg Res 1982;32:65-9.

27. Smith JH, Merrell RC, Raffin TA. Reversal of postoperative anuria by decompressive celiotomy. Arch Intern Med. 1985;145:553-4.

28. Barnes GE, Laine GA, Giam PY et al. Cardiovascular responses to elevation of intra-abdominal hydrostatic pressure. Am J Physiol. 1985;248:R208-13.

29. Caldwell CB, Ricotta JJ. Changes in visceral blood flow with elevated intraabdominal pressure. J Surg Res. 1987;43:14-20.

30. Jacques T, Lee R. Improvement of renal function after relief of raised intra-abdominal pressure due to traumatic retroperitoneal haematoma. Anaesth Intensive Care. 1988;16:478-82.

31. Cullen DJ, Coyle JP, Teplick R, Long MC. Cardiovascular, pulmonary, and renal effects of massively increased intra-abdominal pressure in critically ill patients. Crit Care Med. 1989;17:118-21.

32. Iberti TJ, Lieber CE, Benjamin E. Determination of intra-abdominal pressure using a transurethral bladder catheter: clinical validation of the technique. Anesthesiology. 1989;70:47-50.

33. Schein M, Wittmann DH, Aprahamian CC, Condon RE. The abdominal compartment syndrome: the physiological and clinical consequences of elevated intra-abdominal pressure. J Am Coll Surg. 1995;180:745-53.

34. Burch JM, Moore EE, Moore FA, Franciose R. The abdominal compartment syndrome. Surg Clin North Am. 1996;76:833-42.

35. E.J. Kimball, W. Kim, M.L Cheatham, M.L.N.G. Malbrain. Clinical awareness of intra-abdominal hypertension and abdominal compartment syndrome in 2007.*Acta clinica belgica, 2007; 62-supplement 1.*

36. Saggi BH, Sugerman HJ, Ivatury RR, Bloomfield GL. Abdominal Compartment syndrome. *J Trauma1998;45:597-609.*

37. Wittmann DH. The compartment syndrome of the abdominal cavity. J Intensive Care Med 2000;15:201-20.

38. Malbrain ML, De laet IE. Intra-abdominal hypertension: evolving concepts. *Clin Chest Med.* 2009;30(1):45-70.

39. Cheatham ML.Abdominal compartment syndrome: pathophysiology and definitions.Scand J Trauma Resusc Emerg Med.2009 Mar 2;17:10.

40. Cem Ibis, Aydin Altan. The value of intra-abdominal pressure measurement in patients with acute abdomen. *Asian J Surg* 2009;32(1):33-8.

41. Nathens AB, Brenneman FD, Boulanger BR. The Abdominal Compartment Syndrome. *Can J Surg*1997;4:254.

42. Holzheimer RG, Mannick JA, Abdominal Compartment Syndrome. Surgical Treatment:-Evidence based and problem oriented. Chapter XVI, http://www.freebooks4doctors.com/fb/spec18.htm#surg.

43. J.J. De waele, I. De laet, M.L.N.G. Malbrain rational intra abdominal pressure monitoring: how to do it? *Acta clinica belgica, 2007; 62-supplement 1.*

44. Fietsam R, Villalba M, Glover JL, Clark K. Intra-abdominal compartment syndrome as a complication of ruptured abdominal aortic aneurysm repair. Am Surg. 1989;56:396-402.

45. Pickhardt PJ, Shimony JS, Heiken JP, Buchman TG, Fisher AJ. The abdominal compartment syndrome: CT findings. AJR American Journal of Roentgenology. 1999;173:575-79.

46. Khan S, Verma AK, Ahmad SM, Ahmad R. Analyzing intra-abdominal pressures and outcomes in patients undergoing emergency laparotomy. J Emerg Trauma Shock 2010;3:318-25.

47. Cheatham ML, White MW, Sagraves SG, Johnson JL, Block EF. Abdominal perfusion pressure: a superior parameter in the assessment of intra-abdominal hypertension. J Trauma 2000;49:621-7.

48. Meldrum DR, Moore FA, Moore EE, Franciose RJ, Sauaia A, Burch JM. Prospective characterization and selective management of the abdominal compartment syndrome. Am J Surg 1997;174:667-73.

49. Hong JJ, Cohn SM, Perez JM, Dolich MO, Brown M, McKenney MG. Prospective study of the incidence and outcome of intra-abdominal hypertension and the abdominal compartment syndrome. Br J Surg 2002;89:591-6.

50. Sugrue M, Buist MD, Hourihan F, Deane S, Bauman A, Hillman K. Prospective study of intra-abdominal hypertension and renal function after laparotomy. Br J Surg 1995;82:235-8.

51. Chang MC, Miller PR, Meredeth JW. Effects of abdominal decompression on cardiopulmonary function and visceral perfursion in patients with intra-abdominal hypertension. J Trauma. 1998;440-5.

52. Lazaro G et al.,Haemodynamic effects of increasing intra abdominal pressure in peritoneal dialysis. *Perit Dial Int.*1981;1(4): 41-43.

53. Widergreen, J. T. and Batistella, F. D. The open abdomen: Treatment for intra-abdominal compartment syndrome. J.Trauma. 1994;37(1): 158.

54. David Hopkins, S.W. Gemmell. Intra abdominal hypertension and the abdominal compartment syndrome. British Journal of Anaesthesia 2001;1(2):56-9.

55. Savino JA, Cerabona TD, Agarwal N, Byrne D. Manipulation of ascitic fluid pressure in cirrhotics to optimize hemodynamic and renal function. Ann Surg. 1988;208:504-11.

56. G. Marakis et al.,Alterations In Liver Function Tests Following Laparoscopic Cholecystectomy. *The Internet Journal of Surgery.* 2006; 8(1): ISSN: 1528-8242.

57. Tolga MA et al., Liver Injury in Sepsis and Abdominal Compartment Syndrome in Rats.Surg Today.2006;36(6):519-24.

58. Diebel L et al.,Effect of intra abdominal pressure on abdominal wall blood flow. Am Surg.1992;58(9):573-6.

59. Sugrue M, Jones F, Deane SA, et al. Intra-abdominal hypertension is an independent cause of postoperative renal impairment. Arch Surg 1999;134:1082-5.

60. Scollay JM, de Beaux I, Parks RW. Prospective study of intra-abdominal pressure following major elective abdominal surgery. World J Surg 2009;33:2372-7.

61. J. E. Varela et al. Correlations between intra-abdominal pressure and obesity-related co-morbidities. Surgery for Obesity and Related Diseases 2009;5: 524–8.

62. Wilson A et al. Intra abdominal pressure and the morbidly obese patients: The effect of body mass index. Journal of Trauma – Injury Infection and Critical Care 2010;69(1):78-83.

Annexure 1: Proforma:

OP/IP No. :

DOA:

DOD:

Name :

Age :

Sex :

Weight(kg):

Occupation:

Address :

HISTORY:

Pain in abdomen:

- Duration
- Site
- Nature
- Radiation
- Aggravating Factors
- Relieving Factors

Distension:

Vomiting:

Nature:

Number of episodes:

Quantity:

Fever:

Constipation:

Absolute

Relative

Haematemesis:

Haematochezia:

Malaena:

Trauma:

Others:

Clinical Findings:

Pulse Rate:

Respiratory Rate:

Blood Pressure:

Pallor:

Icterus:

Cyanosis:

Oedema:

Urine Output:

Oxygen Saturation:

Overlying Skin:

Tenderness:

Site:

Guarding:

Rigidity:

Rebound Tenderness:

Organomegaly:

Liver

Kidney

Right: Left:

Spleen:

Free Fluid:

Bowel Sounds:

Abdominal Girth:

Cardiovascular System:

Respiratory System:

Bilateral air entry

Provisional Diagnosis:

Investigations:

Haemogram:

Haemoglobin:

Total Leucocyte Count:

Differential Leucocyte Count:

Erythrocyte Sedimentation Rate:

Liver Function Tests:

Serum Bilirubin:

Total:

Direct:

S.G.P.T:

Serum Alkaline Phosphatase:

Renal Function Tests:

Blood Urea:

Serum Creatinine:

Serum Electrolytes:

Sodium:

Potassium:

X-Ray Chest:

X-Ray Erect Abdomen:

Ultrasonography of abdomen and pelvis:

Diagnosis:

Nature of surgery: Emergency / Elective

Follow up investigations:

Renal function tests:

Liver Function test:

Proforma for daily monitoring:

	POD 0	POD 1	POD 2	POD 3	POD 4	POD 5	POD 6	POD 7
Abdominal Girth								
Pulse								
Blood Pressure								
Intra-abdominal pressure								
Urine Output								
Oxygen Saturation								
Arterial Blood Gas								

POD: Post operative day

Annexure- 2 - MASTER CHART:

S No.	Ip No.	Age / Sex	Diagnosis	Surgery	IAP Grade
1	I10024776	81y/F	Ca.Sigmoid	Sigmoid resection with Hartman's Procedure	0
2	I10026779	63y/F	Pelvic abscess	Laparotomy and proceed	0
3	I10028030	63y/F	Small bowel melanoma	Laparotomy and proceed	0
4	I10028323	40y/M	Ca Stomach	Distal Gastrectomy with GJ	1
5	I10029069	17y/M	Polytrauma with blunt injury abdomen	Laparotomy and proceed, Small bowel resection anastamosis.	2
6	I10027832	51y/M	Retroperitoneal secondaries	Laparotomy and proceed	1
7	I10031432	20y/F	Intestinal obstruction/Abdominal small bowel cocoon	Laparotomy and proceed excision of cocoon sac	0
8	I10027413	45y/M	Duodenal Perforation	Laparotomy with tranquil vagotomy and pyloroplasty	2
9	I10034276	39y/M	RTA with bowel injury	Small intestine resection and anastomosis	1
10	I10034719	20y/M	Perforated peritonitis	Exploratory laparotomy	1
11	I10035621	38y/F	Hereditary spherocytosis	Splenectomy	0
12	I10035921	38y/M	Colonic perforation	Resection anastomosis	0
13	I10036280	52y/M	? Transverse colon diverticular perforation	Resection anastomosis	0
14	I10035437	72y/M	Malignant gastric ulcer	Partial gastrectomy and anastomosis	1
15	I10037162	61y/M	Perforated sigmoid colon with diverticulitis	Resection anastomosis	0
16	I10038523	70y/M	Foreign body aspiration	Laparotomy and proceed	0
17	I10039978	57y/M	Peritonitis	Laparotomy and proceed	0
18	I10040862	67y/F	Strangulated umbilical hernia	Resection anastomosis with mesh repair	0
19	I10041460	62y/M	Liver injury	Laparotomy and proceed	0
20	I10042135	62y/M	Bowel Gangrene	Resection anastomosis	1
21	I10024776	87y/F	Obstructed Inguino femoral hernia	Open mesh repair	0
22	I10042764	41y/M	Blunt Injury Abdomen	Laparotomy and proceed	0
23	I10042710	21y/F	Duodenal Perforation	Resection anastomosis	0
24	I10042627	38y/F	Carcinoma Transverse Colon	Hartmann's colostomy	1
25	I10042800	88y/M	Bleeding Duodenal ulcer	TVGJ	0
26	I10042688	27y/M	GIST	Mesenteric cyst excision	0
27	I10043558	77y/M	Intestinal Perforation	Laparotomy and proceed	1

S No.	Ip No.	Age / Sex	Diagnosis	Surgery	IAP Grade
28	I10043584	45y/M	? Hollow viscous perforation	Laparotomy and proceed	0
29	I10042977	62y/M	HCC	Segmentectomy	0
30	I10043961	45y/F	Duodenal Perforation	Graham's Patch	1
31	I10043947	47y/M	Intestinal obstruction with peritonitis	Resection anastomosis	0
32	I10043584	45y/M	Burst Abdomen	Laparotomy and proceed	0
33	I10043292	43y/M	Splenomegaly	Splenectomy	0
34	I10044372	49y/M	Intestinal Perforation	Resection anastomosis	0
35	I10043292	43y/M	Burst Abdomen	Laparotomy and proceed	1
36	I10045024	87y/M	Gastric Carcinoma	Distal Gastrectomy with GJ	0
37	I10045364	71y/F	Incarcerated Hernia	Resection anastomosis with mesh repair	0
38	I10045095	77y/M	Small bowel stricture	Adhesion release	2
39	I10045910	77y/F	Subacute Intestinal Obstruction	Laparotomy and proceed	0
40	I10046012	76y/M	Small bowel obstruction	Resection anastomosis	0
41	I10045887	52y/M	Carcinoma stomach	Billroth II Gastrectomy	0
42	I10046309	49y/F	Pelvic abscess	Laparotomy and proceed	0
43	I10047003	44y/F	Hollow viscous perforation	Laparotomy and proceed	0
44	I10046515	30y/F	Intestinal obstruction	Resection anastomosis	1
45	I10046012	76y/M	Post operative anastomotic leak	Ileostomy	3
46	I10047448	58y/F	Carcinoma stomach	Palliative GJ	1
47	I10047824	77y/F	Duodenal carcinoma	Palliative GJ	0
48	I10048444	73y/M	Chronic Recurrent Intususception	Right Hemicolectomy	0
49	I10048650	28y/M	?Peritonitis	Laparotomy and proceed	0
50	I10048212	52y/M	Colonic perforation	Ileostomy	1
51	I10048678	20y/M	?Peritonitis	Laparotomy and proceed	0
52	I10048920	52y/F	Intraabdominal fibromatosis	Laparotomy and proceed	0
53	I10048896	42y/F	Carcinoma stomach and pyloric region	Distal Gastrectomy with GJ	0
54	I10049231	82y/F	GIST	Billroth I Gastrectomy	0
55	I10048531	17y/F	Corrosive acid Ingestion - Pyloric Stenosis	GJ	0
56	I10049338	67y/F	Carcinoma stomach	Billroth II Gastrectomy	1

S No.	Ip No.	Age / Sex	Diagnosis	Surgery	IAP Grade
57	I10049979	72y/M	Periampullary Carcinoma	Billroth I Gastrectomy	1
58	I10049745	70y/F	Ischemic bowel	Resection anastomosis	0
59	I10050142	68y/M	Hollow viscous perforation	Laparotomy and proceed	1
60	I10050157	45y/M	Hollow viscous perforation	Laparotomy and proceed	0
61	I10050033	22y/F	Hereditary spherocytosis	Splenectomy	0
62	I10050753	57y/M	Chronic Pancreatitis	Frey's procedure	3
63	I10051625	70y/F	Carcinoma Sigmoid with RCC	Sigmoidectomy with radical nephrectomy	0
64	I10052094	21y/F	Blunt Injury Abdomen	Splenectomy	0
65	I10051544	61y/F	Carcinoma stomach	Total Gastrectomy	1
66	I10052079	32y/M	Splenic abscess	Splenectomy	0
67	I10051887	40y/M	Gangrenous Bowel	Resection anastomosis	0
68	I10052168	35y/M	Duodenal Perforation	Graham's Patch	1
69	I10050915	42y/F	Carcinoma Sigmoid	Left Hemicolectomy	0
70	I10052917	80y/F	Intestinal obstruction	Right Hemicolectomy	0
71	I1000291	40y/M	Perforated Duodenal ulcer	Exploratory laparotomy	2
72	I11001261	44y/F	Perforated Appendicitis	Laparotomy and proceed	3
73	I11001355	51y/F	Intestinal obstruction	Resection anastomosis	1
74	I11003176	36y/M	Pseudopancreatic cyst	Cystogastrostomy	2
75	I11003982	31y/F	Blunt Injury Abdomen	Splenectomy	0
76	I11005685	54y/M	Hollow viscous perforation	Appendicular abscess drainage	2
77	I11006931	30y/F	Blunt Injury Abdomen	Laparotomy and proceed	2
78	I11007984	31y/M	Small bowel injury	Resection anastomosis	1
79	I11009194	40y/M	Blunt Injury Abdomen	Laparotomy and proceed	2
80	I11009281	23y/M	Appendicular perforation	Laparotomy and proceed	3
81	I11009699	71y/M	Intestinal obstruction	Resection anastomosis	1
82	I11010544	23y/M	Splenic Injury	Splenectomy	0
83	I11008500	45y/M	Acute necrotising pancreatitis	Laparotomy and proceed	4
84	I11011484	24y/F	Small bowel obstruction	Resection anastomosis	0
85	I11011828	37y/M	Stab injury abdomen	Exploratory laparotomy	2

S No.	Ip No.	Age / Sex	Diagnosis	Surgery	IAP Grade
86	I11011889	42y/M	Duodenal Perforation	Graham's Patch	2
87	I11008500	45y/M	Acute necrotising pancreatitis	Laparotomy and proceed	4
88	I11014451	57y/M	Internal Hernia	Resection anastomosis	1
89	I11014950	69y/F	Large Bowel Obstruction	Anterior Resection	1
90	I11016034	33y/M	Duodenal Perforation	Graham's Patch	1
91	I11016179	61y/M	Intestinal obstruction	Resection anastomosis	0
92	I11016463	68y/F	Splenic Injury	Splenectomy	0
93	I11016287	33y/F	Carcinoma stomach	Subtotal Gastrectomy	0
94	I110116212	46y/M	Carcinoma stomach	Blllroth II Gastrectomy	0
95	I11017573	56y/M	Perforated Duodenal ulcer	Graham's Patch	0
96	I11017303	37y/F	Mesenteric cyst	Cyst Excision	1
97	I11018865	23y/F	ITP	Splenectomy	0
98	I11019371	68y/M	Ischemic bowel	Resection anastomosis	0
99	I11020454	61y/M	GI Perforation	Left Hemicolectomy	0
100	I11019947	49y/F	Ca. Oesophagus	Ivor lewis procedure	1